"PREVALENCE OF CONGENITAL ANOMALIES OF KIDNEY AND URINARY TRACT (CAKUT) IN CHILDREN: A ONE YEAR CROSS SECTIONAL STUDY AT DR PRABHAKAR KORE HOSPITAL AND MRC BELAGAVI"

BY

REG NO. BM0117002

Dissertation

Submitted to the
KLE Academy of Higher Education and Research,
Belagavi, Karnataka

In Partial Fulfillment
of the requirements for the degree of

M. D. (Doctor of Medicine)

IN

PAEDIATRICS

DEPARTMENT OF PEDIATRICS,
JAWAHARLAL NEHRU MEDICAL COLLEGE
BELAGAVI, KARNATAKA

APRIL - 2020

KLE Academy of Higher Education and Research, Belagavi, Karnataka

Endorsement by the HOD, Principal/Head of the Institution

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Sub: Acceptance Letter

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ABSTRACT

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Congenital anomalies of kidney and urinary tract (CAKUT) are group of abnormalities affecting the kidneys and other structures of the urinary tract. This study was aimed to find the prevalence of CAKUT along with other associated anomalies and to assess the degree of renal dysfunction.

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This one year cross sectional study was conducted in department of paediatrics, KLES Dr Prabhakar Kore Hospital and Medical Research Centre, Belagavi. Newly diagnosed CAKUT cases were enrolled and were evaluated for CAKUT and other associated anomalies and renal dysfunction was evaluated based on Acute Kidney Injury Network's (AKIN's) criteria.

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During the study period there were 7893 admissions in the paediatric ward. Among them 81 cases were diagnosed to have CAKUT but 6 cases (0.07%) were previously diagnosed cases of CAKUT and excluded thereby the diagnosis of CAKUT was confirmed in 75 cases. Hence, the prevalence of CAKUT was 0.95% or 9.5 per 1000 children. Hydronephrosis with pelviureteric junction obstruction PUJO (25.33%) was the common CAKUT followed by Hypospadias (20%). Other associated anomalies were noted in 8% of the children anorectal malformation was the common associated malformation noted among 5.33% of the children. Based on AKIN criteria, stage I renal function was noted in 10.67% of the children and stage II in 9.33% of the children. Majority of study subjects were males (89.33%) and most of

the children belonged to the age group <1 year (57.33%). Gestational diabetes mellitus and pregnancy induced hypertension were the common risk factors noted in 10.67% each. Majority of the children (64%) underwent surgical intervention and majority of the children improved (69.33%).

Conclusion and interpretation

Prevalence of CAKUT in children aged from one month to 18 years is 9.5 per 1000 children. Hydronephrosis is the common anomaly due to pelvic ureteric junction obstruction and hypospadias.

Keywords

Congenital anomalies of kidney and urinary tract; Hydronephrosis; Renal dysfunction;

CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

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Declaration by the Candidate

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Date:

Place: Belagavi. **Dr. MANOJ KADLIMATTI**

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It is an immense pleasure for me on this occasion, to convey my gratitude and regards to all the personalities to whom I owe a lot.

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I offer special thanks to **Dr. (Mrs) N. S. Mahantshetti** **MD, Principal,** Department of pediatrics, Jawaharlal Nehru Medical College, Belgaum for her valuable suggestions and the help offered during this study. Her enthusiastic support, valuable suggestions, unwavering faith and co-operation has continually motivated me.

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Metgud MD, Dr.Abhilasha Sampagar MD, Dr.Bhavana Koppad MD ,Dr. Meenakshi B.R MD, Dr. Meenakshi.S MD and **Dr. Sindhura B.M MD,** and **Dr.Govind MD** for their valuable suggestions and encouragement.

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Finally I thank **God Almighty**, for making all these wonderful people happen to me and pray him for continued blessing and success.

Date:

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LIST OF FIGURES

INTRODUCTION

Congenital anomalies of kidney and urinary tract (CAKUT) are group of urinary systems anomalies. In spite of the abnormality may not become evident until later in life, it is present since birth. They results from deviation of the normal development of the urinary system.[1] Ureter , urinary bladder and urethra are the other parts of the urinary tract that may be affected.[1] They represents 20% to 50% of all fetal congenital anomalies,[2-5] and 30% to 60% of cases may lead to childhood-onset chronic kidney disease (CKD)[6]. These are some causes of renal replacement therapy (RRT) and premature mortality in the young adult population.[2,7,8]

Some studies have suggested that, 5% to 6.1% of developmental defects in newborns attributed to gestational DM.[2,9] A recent study suggested that, diabetes during the first 20 weeks of gestation involve more risk.[10] The probable causes of congenital urinary system developmental defects are composite and involve health of the mother,[9] hereditary, and environment in the womb.[2]

The complete range of developmental defects of urinary system includes, mild forms, presents asymptomatically like double ureter or minimal ureteral pelvic obstructions, to severe renal agenesis which is bilateral and renal dysplasia.[11] Developmental defects of urinary system associated with syndrome will have additional developmental defects outside of the urinary system. Whereas isolated developmental defects of urinary system are present in non-syndromic conditions.[12]

18–20 weeks of gestation is crucial for antenatal ultrasonography where many developmental defects can be diagnosed. Presence of single umbilical artery, difficulty in feeding, reduced urine output, abdominal wall musculature deficiency, and abdominal mass, undescended testes or multiorgan developmental defects are the postnatal manifestations.[13]

3–6 per 1000 live births is the Prevalence of developmental defects of urinary system. Children with chronic irreversible renal failure have 30 times lower survival rate. Newer methods are needed to avert the urinary system anomalies, protect its function, and narrow down the associated cardiac and vascular diseases.[12] Considering these facts, also, most of the available data on epidemiology is from western population and little or no data is available in Indian population. The present study was planned to ascertain the frequency of CAKUT in children till 18 years of age and other associated anomalies and to assess the renal dysfunction according to AKIN's criteria.

OBJECTIVES

The objectives of this study were;

Primary

1. To study the prevalence of congenital anomalies of kidney and urinary tract(CAKUT) in children admitted in KLES DR Prabhakar Kore Hospital.

Secondary

1. To assess the degree of renal dysfunction by AKIN criteria.
2. To study the other associated anomalies.

REVIEW OF LITERATURE

Congenital anomalies of the kidney and urinary tract

The developmental defects of the urinary systems are highly frequent in the general population, most common being the urinary tract dilations. Without the thorough knowledge about the healthy development of kidney and urinary tract the understanding of these anomalies is difficult.[14]

Development of the urinary system

Healthy development of the kidneys

Kidney has two sites of origin. The nephrons which are excretory tubules originated from lower part of the nephrogenic cord called as metanephros. The lower part of mesonephric duct gives raise to a diverticulum called ureteric bud from which the collecting part originates. The growth of kidney has three stages i.e pronephros in fishes it is normal functioning kidney. This pronephros succeed into the next stage by mesonephros. Mesonephros is the functioning kidney in some fishes.[14]

The pronephros is formed in upper region of nephrogenic cord and appearance of mesonephros in middle region and formation of metanephros in sacral region. These are nonfunctional in human and they disappears immediately after its formation. A nephric duct formed, it persists by ending in cloaca. The mesonephros consists of excretory tubules develop into middle region, they drain into nephric duct which are called as mesonephric duct. Most of these tubules disappear, some are take part in testis formation.[14]

Ureteric bud dilated to form an ampulla. Division of the ampulla takes place, the three to five divisions fuse and form pelvis of the kidney. Major and minor calyces and collecting tubules are formed later. The nephron is formed by differentiation of cells of metanephric blastema induced by ampulla. These cells form a solid mass in reference to ampulla and they turn into a vesicle. The vesical will turn into S shaped tube and the tuft of capillaries invade this tube at the distal end and forms a glomerulus.[14]

Ascent of the kidney

The metanephros leads formation of human kidney which are present in groin region. The upward movement of the kidney to loin region is caused by growth of embryo and abdominal wall growth. If the right and left umbilical arteries come in between during this process, the ascent of the kidney will be affected.[14]

Rotation of the kidney

At first the normal hilum of the kidney faces anteriorly and it comes to face medially by gradual rotation.[14]

Absorption of lower parts of mesonephric duct into cloaca

Urogenital sinus is formed by opening of lower end of mesonephric duct into the part of cloaca. The vesicourethral canal absorbs mesonephric duct, so that there is separate openings into cloaca for the ureteric bud and mesonephric duct.[14]

Development of ureter

Ureter lies between vesicourethral canal and the pelvis which is originated from ureteric bud.[14]

Development of the urinary bladder

The upper part of the vesicourethral canal gives raise to epithelium of urinary bladder and the mesonephric ducts which is absorbed gives raise to epithelium of the trigone. The splanchnopleuric mesoderm gives raise to the muscular and serous walls of the organ.[14]

Development of the female urethra

The lower part of the vesicourethral canal gives raise to female urethra. Mesonephric duct also forms the posterior wall of this canal so it is also mesodermal in origin. Urogenital sinus especially the pelvic part also contributes to the female urethra.[14]

Development of the male urethra

The male urethra near the bladder till the openings of ejaculatory ducts is originated from lower part of vesicourethral canal (endoderm). The Absorbed part of mesonephric forms the posterior wall of this part. The urogenital sinus (pelvic part) forms the prostatic and membranous urethra. penile urethra which is present in shaft of penis is originated from urogenital sinus (the phallic part). The glans of penis contains terminal part which is originated from ectoderm.[14]

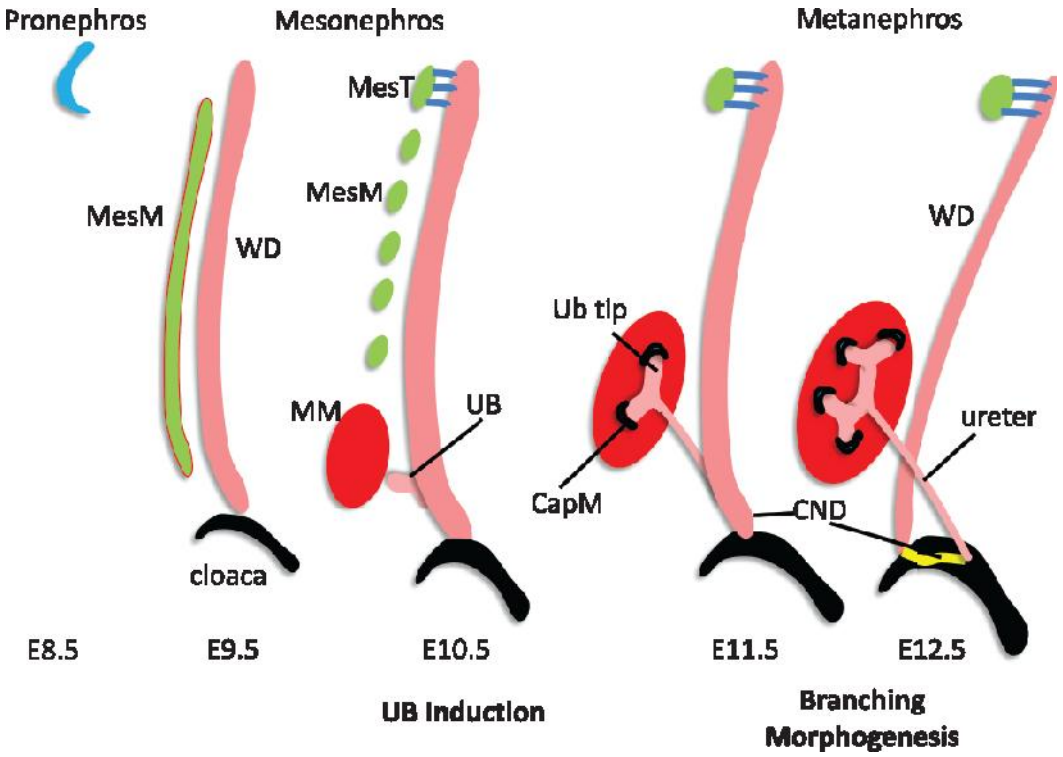


Figure 1. Mammalian kidney developmental stages[15]

Pathological and clinical features of developmental defects of urinary system

CAKUT comprises a wide range of urinary system anomalies, consists of agenesis of kidney and hypoplasia, duplication and position defects in the lower urinary system. In this review most emphasis is on the urinary systems anomalies and their clinicopathological features.

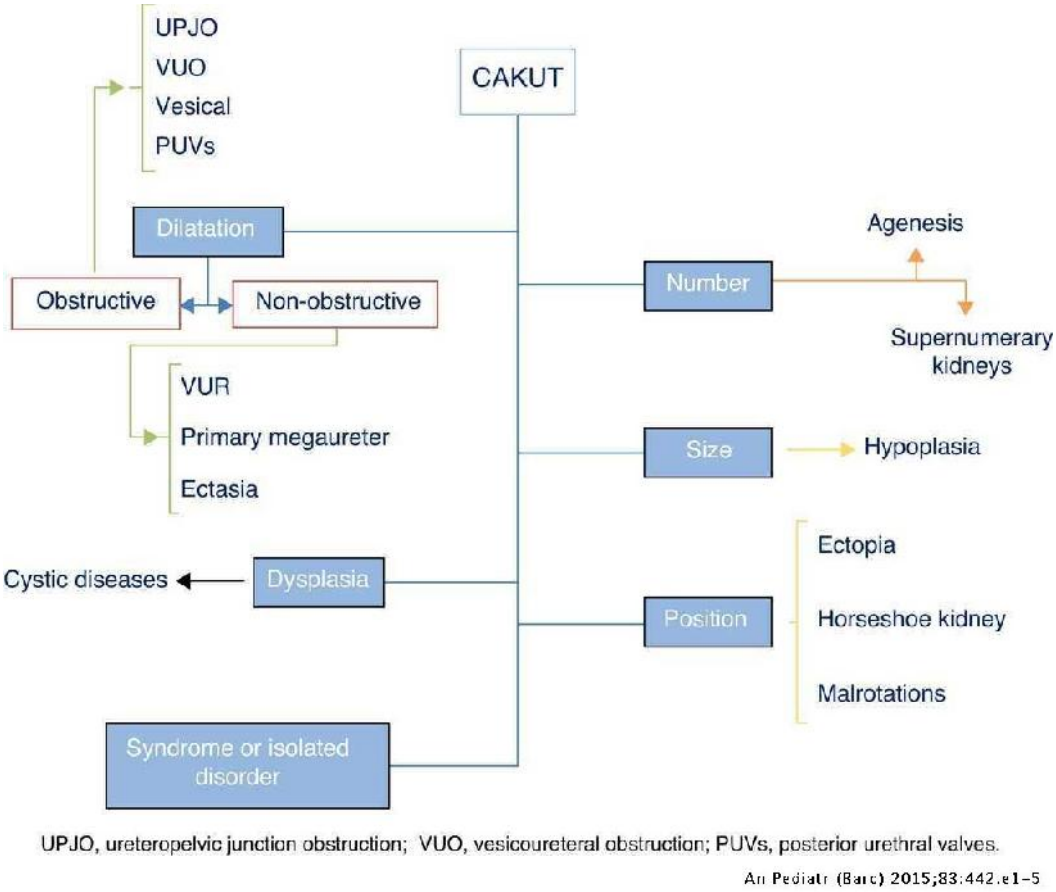


Figure 2. Pathway of developmental defects of urinary system.[16]

Duplication and supernumerary kidneys

In supernumerary kidneys, there is emergence of multiple ureteric buds and they grow into the metanephric mesenchyme.[16-18] Bifurcation of the ureteric bud stalk before invading or before the beginning of the branching of primary ureteric bud stalk in the metanephric mesenchyme causes partial duplication, which are insignificant. In complete duplication, emergence of two ureteric buds from the Wolffian duct that results in complete excretory organs and ureteral tubes that may separately inserted into the vesica (Figure 3). As their development is from the same metanephric mesenchyme they look fused. In majority of such cases, tunneling of the ureter is abnormal, leading to reflux, but the location of entering of lower ureter into the

bladder is normal.[19] Ureterocele will occur when the superior ureter lodge more inferiorly, near the organs of reproduction or in the urethra. The ureterocele often drains the upper kidney or the upper pole of a duplex kidney. Dysplasia or obstructive nephropathy is caused by obstruction or reflux. High blood pressure, pain and chronic irreversible renal failure are the presenting symptoms.[17] Women are commonly affected in duplication.[17]

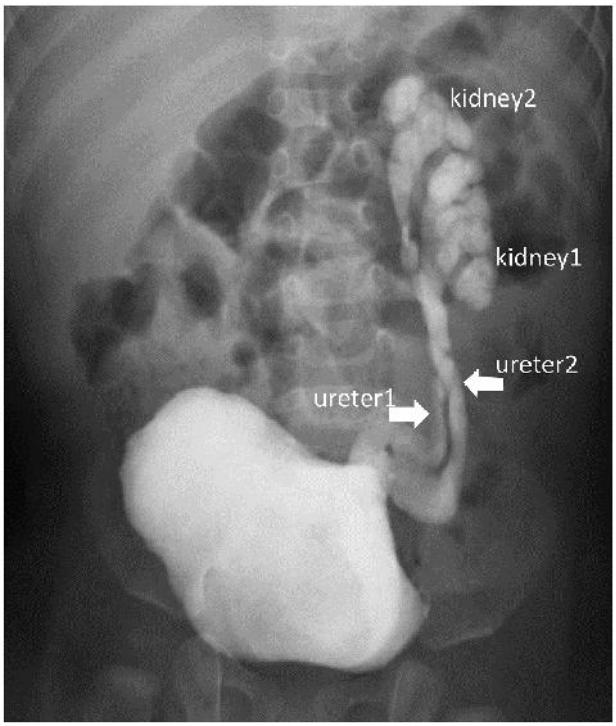


Figure 3. MCU picture showing urinary system duplication. The primary ureter (ureter1) occupies the position above in the bladder empties the lower kidney (kidney1). The enlarged ureter2 occupies the position below in the bladder empties the upper kidney (kidney2).[16]

Agenesis

Defective/Delayed Wolffian duct growth or ureteric bud induction causes agenesis of the kidneys. It may involve one kidney or both. If it involve bilaterally life is incompatible and more commonly affects males.[20] Oligohydramnios and abnormal lung development are the result of kidney agenesis during embryogenesis.[21] If it involves one kidney the opposite kidney is hypertrophied and while routine sonography it can be detected. With no gender differentiation it occurs more on the left. Dysplasia, ectopia, reflux and proteinuria are the other anomalies associated with unilateral agenesis.[16]

Hypoplasia

In Hypoplasia there is reduced branching morphogenesis that leads to decrease in number of nephrons. There is no destruction of kidney architecture, cortex and medulla are normally organized. Incidence is 1/1000 in unilateral cases, occurrence of bilateral cases are less (1/4000). In bilateral cases, they develop chronic irreversible renal failure in children with hypertension.[20] There may be no consequence in simple unilateral hypoplasia. Renal artery hypoplasia may be associated with hypoplasia.[16]

Dysplasia

Architectural disorganization, nephrons immaturity, branching defects, undifferentiated stroma are the characteristic features of dysplastic kidneys.[22] There is a loss of correlative interactions among the ureteric bud, metanephric mesenchyme and stroma during branching morphogenesis. Dysplastic kidneys show cystic changes, size can be normal, or bulkier, or smaller. Dysplasia can be unilateral or present on both sides.[20,22] Histological examination of the tissue is the definitive diagnosis, the

clinical diagnosis is made amid by antenatal ultrasound. 50–70% of the patients may have both affected kidney in unilateral kidney. In patients with well controlled hypertension and infection the prognosis in unilateral dysplasia is good. High degree of dysplasia or branching arrest leads to complications of the kidney or unformed kidney. Renal cysts and the lack of a healthy pelvicalyceal system are the characteristic features of abnormal metanephric differentiation causing multicystic dysplasia. They may undergo automatic involution, increase in size or persist without any change.[16]

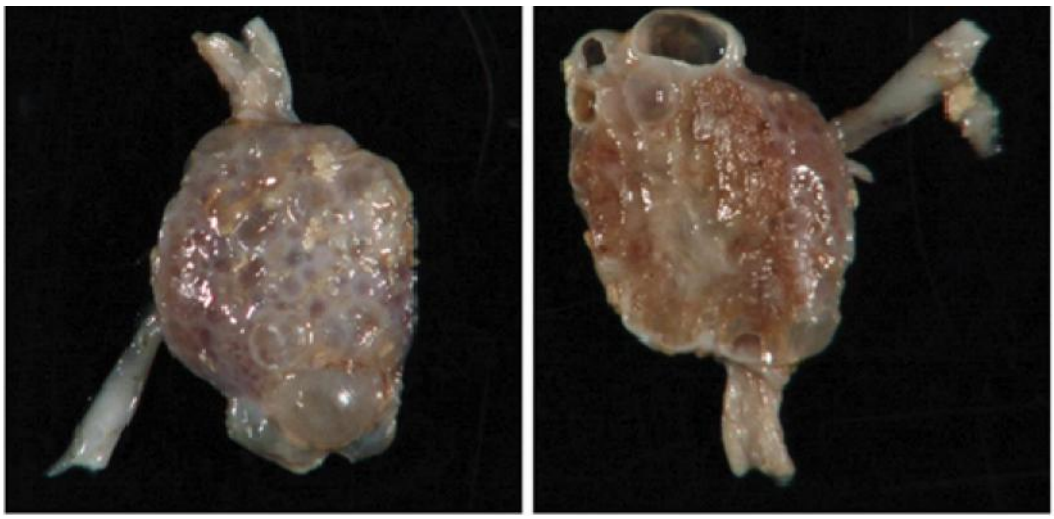


Figure 4. Renal dysplasia. (A**) Cystic dysplastic kidneys gross morphology. left picture shows the uneven shape of the whole kidney and the cut surface picture on the right shows multiple cysts and no organization.**[16]

Position defects (horseshoe kidneys, renal ectopia and malrotation)

Horseshoe kidneys

It is the abnormal fusion of the kidney. Here the mesenchyme or the Wolffian ducts are positioned abnormally, affecting the inclination of the renals. The parenchyma may have normal cytology, the associated reflux or obstruction during development may cause destruction of parenchyma.[16]

Crossed fused renal ectopia

It is the position defect of the renal system and is second most common, here the kidney has fused with the opposite kidney and crossed the midline. Smaller in size compared to normally placed kidney.[16]

Ectopia

Ectopic kidneys are situated away from the renal fossa. Kidneys present ipsilateral to the pelvis are called simple ectopic kidney or they can crossed to opposite side are called crossed ectopic kidneys (opposite to the entry site of ureter into the vesica).[23] In pelvic horseshoe kidneys, ectopia and fusion occurs together.[16]

Malrotation

Here in this condition the hilum of the kidney will be faced anteriorly. There is absence of rotation towards medially during the inclination which leads to abnormal rotation along their long axis. They usually are asymptomatic they present with some other anomalies.[16]

Hydronephrosis

Interruption for the movement of urine from the kidney causes enlarged and tortuous renal pelvis and calyces leads to hydronephrosis.[24] kidney defects that affect the urine concentration, leading to polyuria that oppress the peristalsis of the pyelo-ureter, causing interruption of urine excretion. kidney stones caused by cystinuria causes obstruction, by blocking urine outflow and affecting ureteral peristalsis. Thus hydronephrosis is not a separate disease per se, the diseases affecting the lower urinary system directly and indirectly causes it.[16]

Obstructive nephropathy and obstructive uropathy

Tubular atrophy, inflammation and fibrosis are results of persistent hydronephrosis.[25-27] These are pathological changes known as 'obstructive nephropathy' and 'obstructive uropathy'. These terms have similar meanings, pathological changes within the kidney is obstructive nephropathy and with involvement of the lower urinary tract is obstructive uropathy. Chronic kidney disease is not uncommon in obstructive nephropathy. Radial dilatation of the tubules and ducts as a result of elevated hydrostatic pressure associated with obstruction which causes increased apoptosis of epithelium and atrophy of tubules which in turn causes a decrease in the filtration from the glomerulus. As the obstructive nephropathy progresses it causes infiltration of immune cells.[28-30]

Early intervention to preserve the kidney function is critical, by relieving the blockade of the urinary tract but is not common. In clinical setting partial obstruction is more common and when both sides involved it may progress to obstructive nephropathy, chronic irreversible renal failure and death.[16]

Ureteropelvic junction obstruction

It is the cause of congenital obstructive nephropathy by obstructing the flow of urine through the UPJ, that causes renal damage (Figure 5).[31,32] Ultrasound detects upper urinary tract dilation in 1/100 pregnancies. Hydronephrosis which is antenatally detected, nearly 50% are caused by UPJ obstruction. They are transient, but some remains significant clinically. More common in males and left side. Blood vessels crossing this junction also cause obstruction at the ureteropelvic junction (UPJO).[16]

Obstruction at the ureteropelvic junction is due to intrinsic causes, surgery remains the only option, in which resected and anastomosis is done (Anderson–Hynes dismembered pyeloplasty).[16]

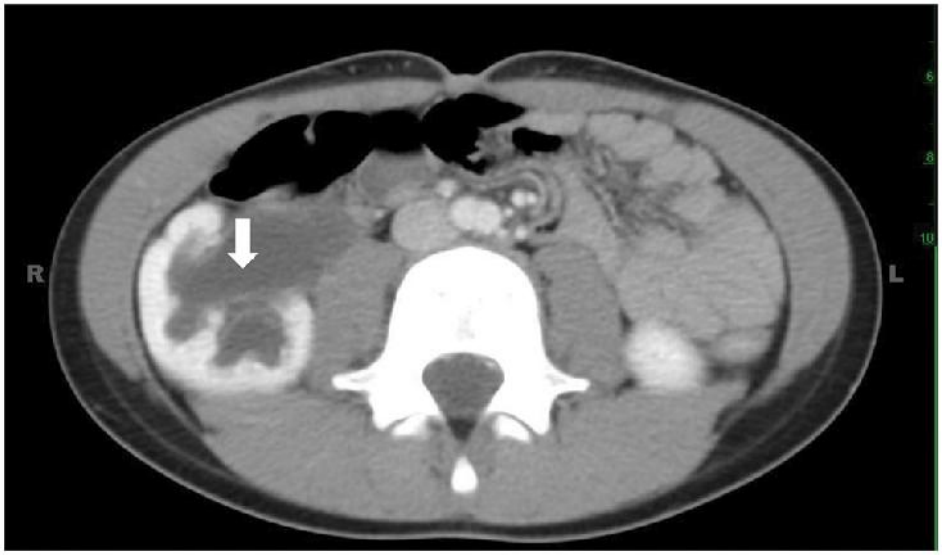


Figure 5. Hydronephrosis and obstruction. The radiographic image shows, due to lower urinary tract obstruction there is diffuse dilation of the collecting system with dilated pelvis (arrow).[16]

Vesicoureteral reflux

Vesicoureteral reflux (VUR) is the movement of urine from the vesica into the tube connecting kidney and the urinary bladder as against normalcy.[33] A voiding cystourethrogram (VCUG) where there is retrograde flow of radio-opaque contrast material is diagnostic modality of VUR (Figure 6).[34,35] VUR patients with advanced stages develop reflexive nephropathy, similar to obstructive nephropathic pathology. Reflexive nephropathy defers pathophysiologically from obstructive nephropathy as scarring of parenchyma of kidney occurs in former because of impact of reflex damage to kidney. The compound papillae in these regions with a round opening that creates smooth path for the retrograde flow. Primary VUR is isolated urinary tract anomaly. Urinary bladder disorder caused by a lesion in the nervous system, congenitally patent valves in posterior urethra (PUV) or ureterocele are associated with VUR.[36-38] Cause of UTI in setting of VUR is primarily due to migration of bacterial inhabitants from bladder to ureter causing scarring and permanent damage.[39]

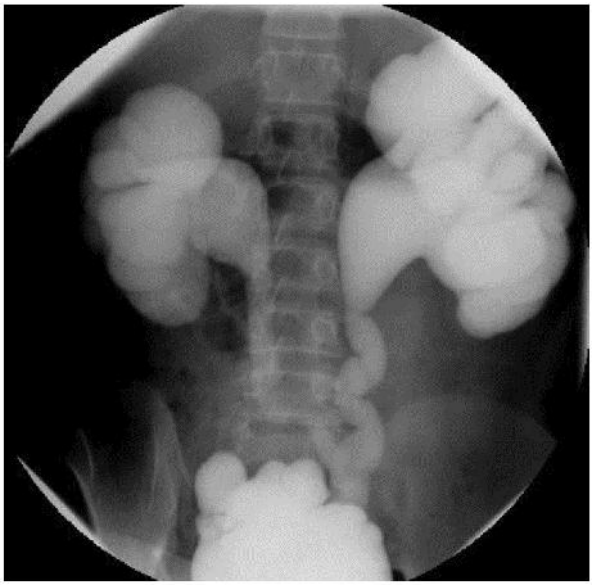


Figure 6. Vesicoureteral reflux (VUR). Vesicouretero cystogram shows high-grade (Grade 5) VUR. Note the dilated, tortuous ureter, with reflux involving the kidney parenchyma, highlighted by the white dye almost spanning each kidney.[16]

Ureterovesical junction obstruction and hydroureter

The normal site of bladder which receives the ureter is base of the bladder is called ureterovesical junction. Enlargement of the ureter (hydroureter or megaureter) and finally stagnation of urine in kidney, is caused by pressure exerted by the urine in backward manner, associated with obstruction at the junction of ureter and vesica. Persistent UVJ obstruction will cause kidney damage similar to UPJ obstruction, but it is less common cause. A poor distal ureteral segment peristalsis causes UVJO, abnormal insertion at the vesica, a short segment of ureter at inner surface of vesica, infection, tissue scarring, kidney stones are other causes. The obstruction can be relieved by resecting the involved pathological segment and anastomosing the ends.[16]

Ureterocele, ectopic ureter and duplicated ureter

Ureterocele is a sac like dilatation of the end part of the ureter's inner surface of the vesica (Figure 7) [176, 199].[24,40] Occasionally they cause urinary tract obstruction which in turn causes kidney damage otherwise they are asymptomatic. Kinking and reflux prevented by the bladder trigone, which helps the intravesical segment of the ureter for the same. The normal site of bladder which receives the ureter is base of the bladder. An aberrant entry of ureter into the bladder and angle will compromise the function of the trigone which causes reflux or obstruction. In the unfortunate event fistulous communications can be created when lower ureteral tube connects to organs of reproduction[41-44] causing obstruction, leading to stagnation of urine in ureter and kidney and renal injury.

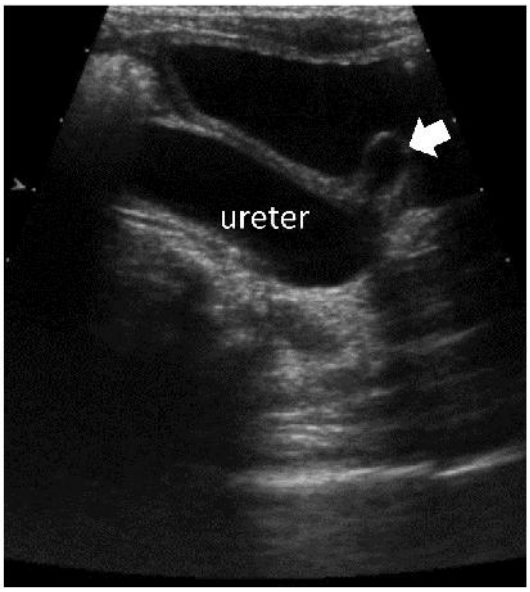


Figure 7. Ureterocele. ultrasonography picture showing a ureterocele (arrow) in pelvis and abdomen. Note the clear blind ending ureterocele lining (arrow) connected to the dilated ureter (long hypoechogenic tube).[16]

Bladder outlet obstruction and posterior urethral valves

It is the hindrance to the movement of urine from the vesica into the urethral tube. hyperplasia of prostate, tumor present in the bladder, stones present in the bladder, congenital patent valves in posterior urethra, are common causes. Congenitally patent PUV may be acknowledged as the commonest cause in male infants and the reason for hydronephrosis picked up in prenatal sonography.[45-47] Bilateral kidney damage which leads to kidney failure and eventually causes mortality. Prenatal ultrasound is the screening method to detect and diagnosed after birth by the same. Bilateral hydroureter/hydronephrosis are common associations of PUV characterized by uneven surface of the bladder wall and 'keyhole' sign in the bladder neck (Figure 10). Presence of the 'valves' or 'membranes' can be demonstrated by VCUG and/or cystoscopic evaluation. Immediate relief is provided bladder catheter but the endoscopic ablation is the corrective procedure.[16]

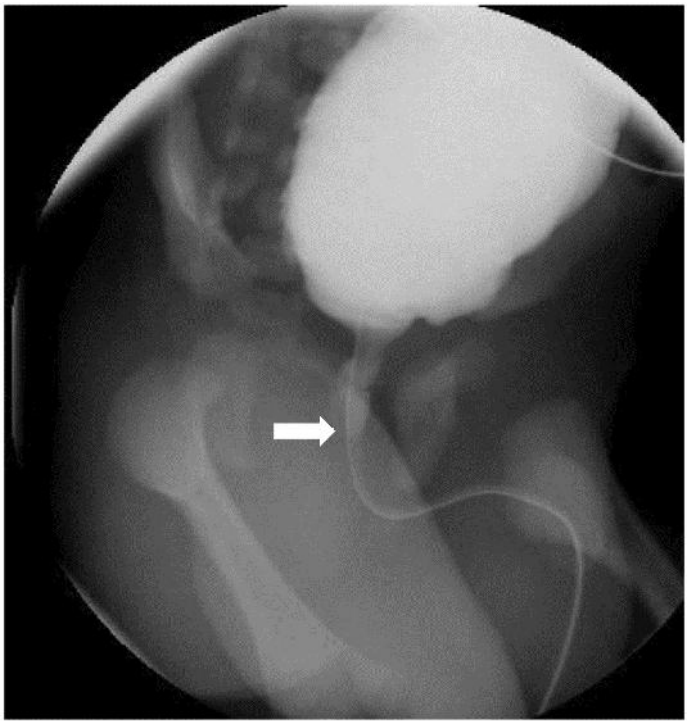


Figure 8. Posterior urethral valves (PUV). very slender urethra (arrow) due to congenital patent valves in posterior urethra can be seen, that lead to enlarged bladder with uneven surface[16]

Recent studies on prevalence of developmental defects of urinary system

In 2016, a prospective study done by Karambelkar GR et al.[48] reported the usefulness of radiological screening and biochemical evaluation can be employed in babies with family history of CAKUT and increased risk factors. Hydronephrosis being the commonest finding in antenatal and postnatal ultrasonography has a significant resolution. Early identification of CAKUT anomalies by prenatal screening prevent or slow end stage renal disease (ESRD) by possible early surgical or medical intervention.[R10]

Wills V. et al.[49] reported a study in 2017 from Kerala India. Authors commented that, the anomalies were found to involve the genitourinary system and

the cardiovascular system in comparison to the others. The major risk factor identified was maternal Diabetes. Emphasis has been made on prevention by propagating awareness during adolescents, counselling prior to conception and screening antenatally. Availability of Pediatric surgery and Rehabilitative facilities to improve the quality of life would be warranted.

In the year 2016, study was reported by Tain YL et al.[2] in Taiwan. 4.2 per 10,000 live births was the incidence of CAKUT. They found that congenital anomalies were more common in, mother with gestational diabetes, thalassemia/hemochromatosis, antenatal polyhydramnios or oligohydramnios. Preterm delivery, low birth weight and male baby were probable to have congenital anomalies.

Gong YL et al.[50] in 2018 reported a study to establish ultrasound as a screening method to detect the developmental defects of the urinary systems in neonates. The reference point for anterior–posterior renal pelvic diameter (APRPD) developing features of uropathy secondary to obstruction was studied which revealed ultrasound as an effective tool of screening for early detection in patients with high grade renal pelvic diameter (RPD), renal and urinary abnormality in whom intervention can be done at early stages.

In (2019) Li ZY et al.[51] in Zhejiang Province, China. Studied the epidemiology of CAKUTs. 1.60 per 1000 livebirths was the prevalence of developmental defects of urinary systems. More common in male, multiple births and are more common in urban population. Most common associated with anomalies of the heart. Hydronephrosis was the main subgroups of anomaly.

MATERIAL AND METHODS

This study was done from January 2018 to December 2018 in the Department of Pediatrics, KLES charitable Hospital and Medical Research Centre, Belagavi.

Study design

The study design was a hospital based cross-sectional study.

Study duration and period

This study was carried out for the period of one year from January 2018 to December 2018.

Place

This study was conducted in the Department of Pediatrics KLES Charitable Hospital and Medical Research Centre, Belagavi a tertiary care teaching hospital attached to Jawaharlal Nehru Medical College, Belagavi.

Source of data

This study was conducted among the children admitted under the Department of Pediatrics, KLES Charitable Hospital and Medical Research Centre, Belagavi

Sample size

The minimum effect size to achieve study objectives was 60 cases. However, a total of 75 cases fulfilled the selection criteria and were enrolled.

Sampling size calculation

The sample size was calculated from the formula as mentioned below.

$n=4*p\ q\ /\ d^2$

Where,

n = sample number

p = Prevalence of the disease according to the previous study[48]

Q = 100-p

D = 2 (Precision)

Therefore,

$n = 4 * 0.6*99.4\ /2^2 = 59.64 \approx 60$

According to this formula the minimum effect size to estimate prevalence of CAKUT was 60 cases. However, a total of 75 cases fulfilled the selection criteria. Hence 75 cases were enrolled.

Selection Criteria

Inclusion Criteria

- Newly diagnosed cases of CAKUT in the age group from birth to 18 years of age during the study period.

Exclusion criteria

- Previously diagnosed congenital anomalies of kidney and urinary tract.

Ethical clearance

At the beginning, Ethical and Research Committee approval taken.

Informed Consent

The parents of children fulfilling selection criteria were briefed about the nature of the study and a written informed consent was obtained from parents / caregivers to participate in the study prior to the enrolment (Annexure I).

Method of collection of data

Parents of the children with CAKUT who fulfilled the selection criteria were interviewed and demographic data including age, gender and socio economic status along with chief complaints. Also past history, treatment history and antenatal history (with special emphasis of the second and third trimester scan), gestational age and history of marriage in blood relation were noted. Risk factors in mothers such as hypertension and diabetes mellitus during pregnancy, were noted. Further these children were subjected to detailed physical examination and were evaluated for vitals and anthropometry. Head to examination was done followed by systemic examination. Also children were examined for clinical signs and symptoms with special emphasis for CAKUT.

Investigations

The selected children underwent following investigations.

- Serum creatinine
- Blood urea nitrogen
- Urine examination – Urine routine, urine specific gravity, urine microscopy.

- Micturating cystourethrography (MCU)
- Magnetic resonance (MR) urography
- Renal nuclear scan (dimercaptosuccinic acid [DMSA]/ diethylene triamine Penta acetic acid [DTPA]).

Outcome variables

Diagnosis of CAKUT

Children were evaluated for diagnosis of CAKUT. The clinical diagnosis of CAKUT was based on clinical presentation and history. The final diagnosis of CAKUT was done based on the clinical examination as well as investigations including imaging.

Renal dysfunction

The renal dysfunction was evaluated based on AKIN criteria as below.

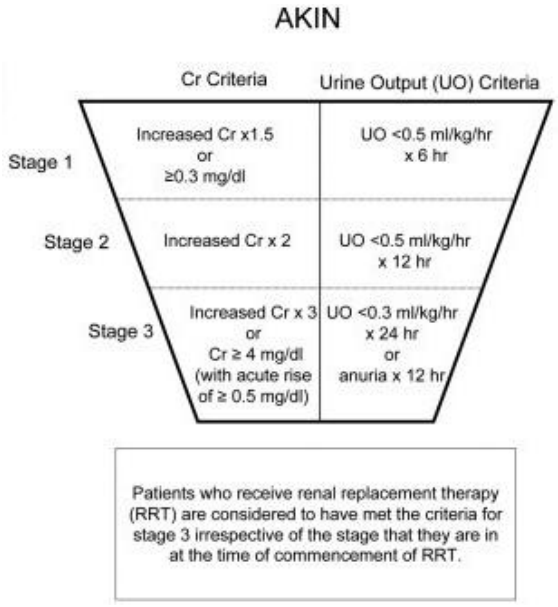


Figure 9. Acute Kidney Injury Network (AKIN) classifications for acute kidney injury[50]

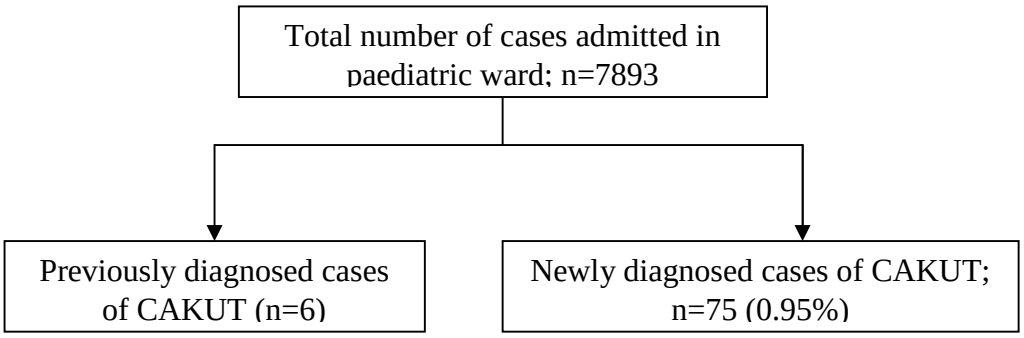
Statistical analysis

The data was coded and tabulated on excel spreadsheet and master chart was prepared (Annexure III). The data was analysed using SPSS version 20.0 statistical software. The categorical data was expressed in terms of rates, ratios and percentages and the continuous data was expressed as mean ± standard deviation.

RESULTS

The present hospital based cross-sectional study for the period of one year from January 2018 to December 2018 was done among the children admitted under the Department of Paediatrics, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. The CONSORT diagram for enrolment and CAKUT is as shown below.

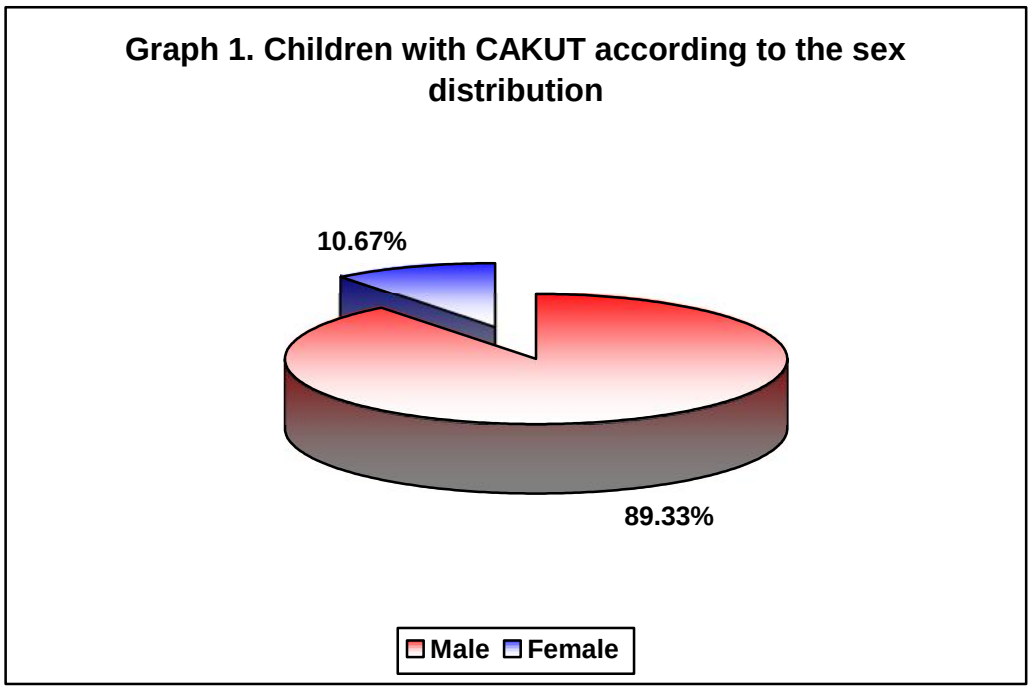
Figure 10. CONSORT diagram for enrolment and prevalence of CAKUT



During the study period there were 7893 admission in the paediatric ward. Among them 81 cases were diagnosed to have CAKUT. Of them, 6 cases (0.07%) were previously diagnosed cases of CAKUT. Hence prevalence of CAKUT was 0.95%

Table 1. Children with CAKUT according to the sex distribution

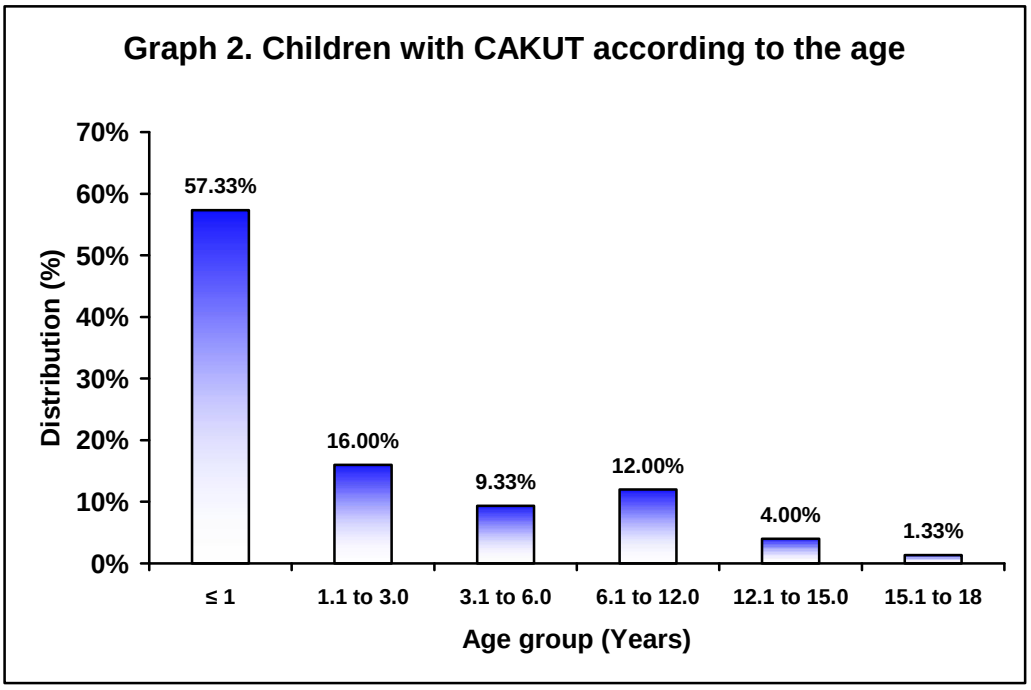
Sex	Distribution (n=75)	
	Number	**Percentage**
Male	67	89.33
Female	8	10.67
Total	**75**	**100.00**



In the present study 89.33% of the cases with CAKUT were diagnosed among males while10.67% of the cases were diagnosed among females.

Table 2. Children with CAKUT according to their age

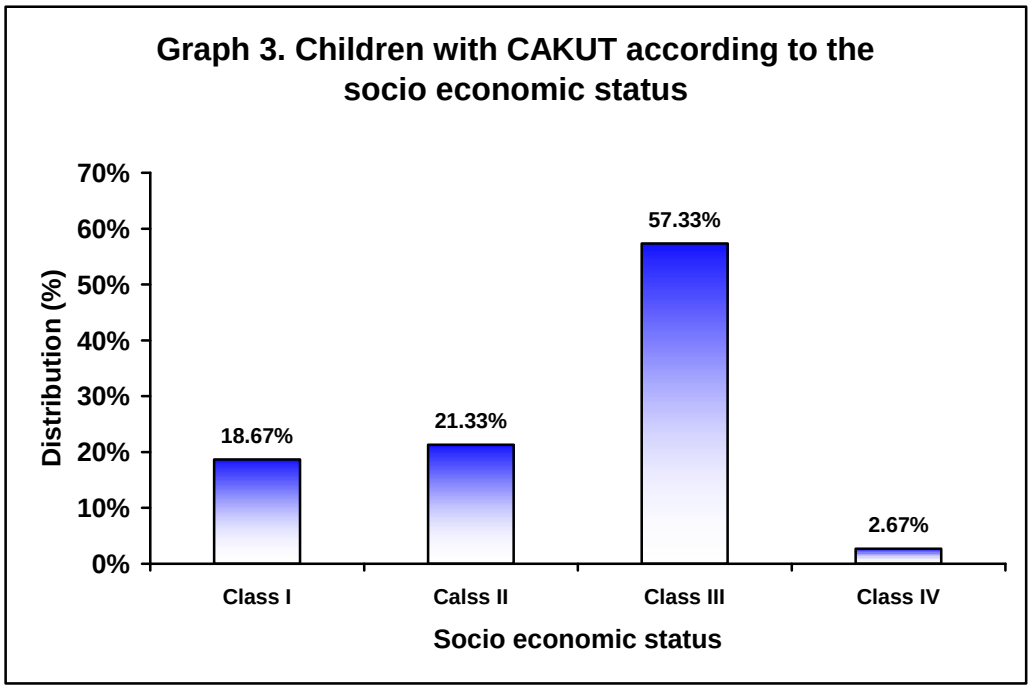
Age group (years)	Distribution (n=75)	
	Number	**Percentage**
≤ 1	43	57.33
1.1 to 3.0	12	16.00
3.1 to 6.0	7	9.33
6.1 to 12.0	9	12.00
12.1 to 15	3	4.00
15.1 to 18	1	1.33
Total	**75**	**100.00**



In this study the age of the children with CAKUT ranged between three days to 18 years. The mean age was 2.83±4.11 and the median age was 0.91 years. Most of the CAKUT cases were aged less than or equal to one year (57.33%).

Table 3. Children with CAKUT according to the socio economic status

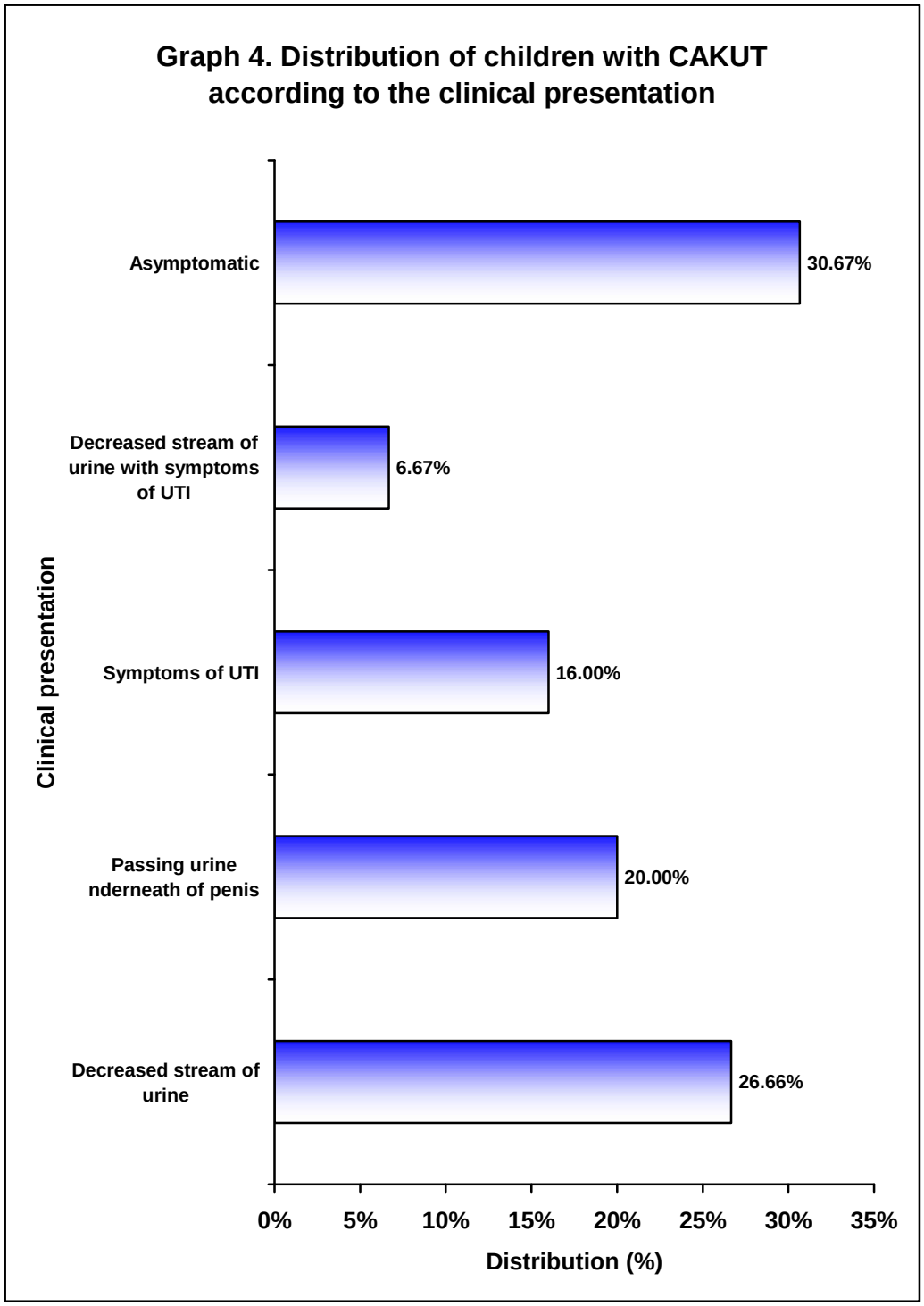
Socio economic status	Distribution (n=75)	
	Number	**Percentage**
Class I	14	18.67
Class II	16	21.33
Class III	43	57.33
Class IV	2	2.67
Total	**75**	**100.00**



In the present study most of the cases with CAKUT belonged to socio economic class III (57.33%) according to the Modified B. G. Prasad's classification.

Table 4. Distribution of children with CAKUT according to the clinical presentation

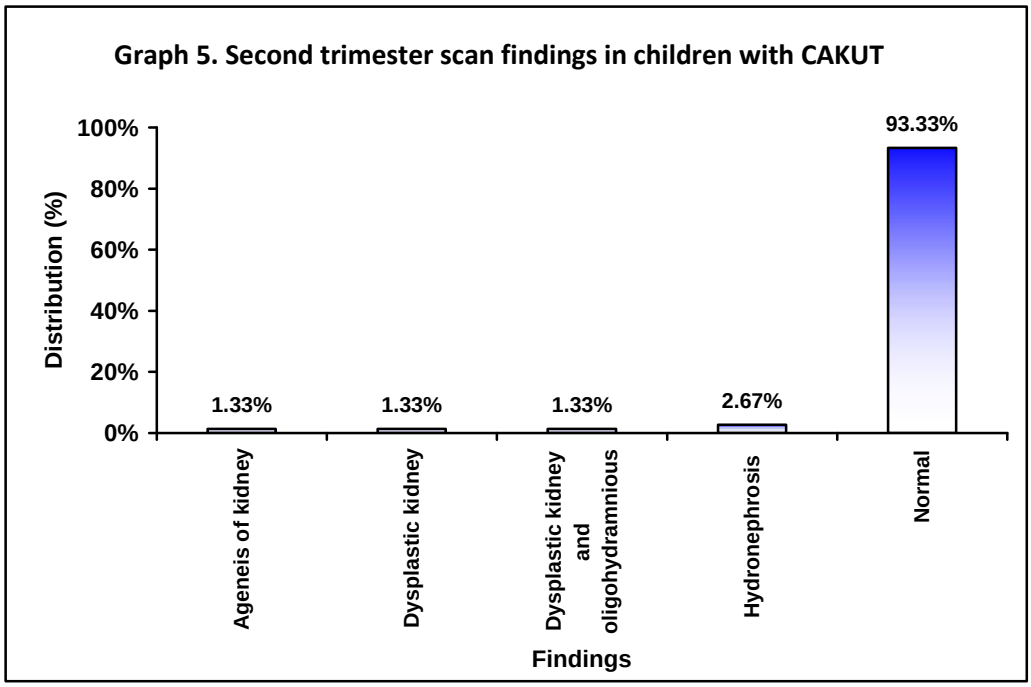
Clinical presentation	Distribution (n=75)	
	Number	**Percentage**
Decreased stream of urine	20	26.66
Passing urine underneath of penis	15	20
Symptoms of urinary tract infection (UTI)	12	16.00
Decreased stream of urine with symptoms of UTI	5	6.67
Asymptomatic	23	30.67
Total	**75**	**100.00**



In this study most of the cases presented with decreased stream of urine (26.66%) while 30.67% of the cases were asymptomatic.

Table 5. Second trimester scan findings in CAKUT.

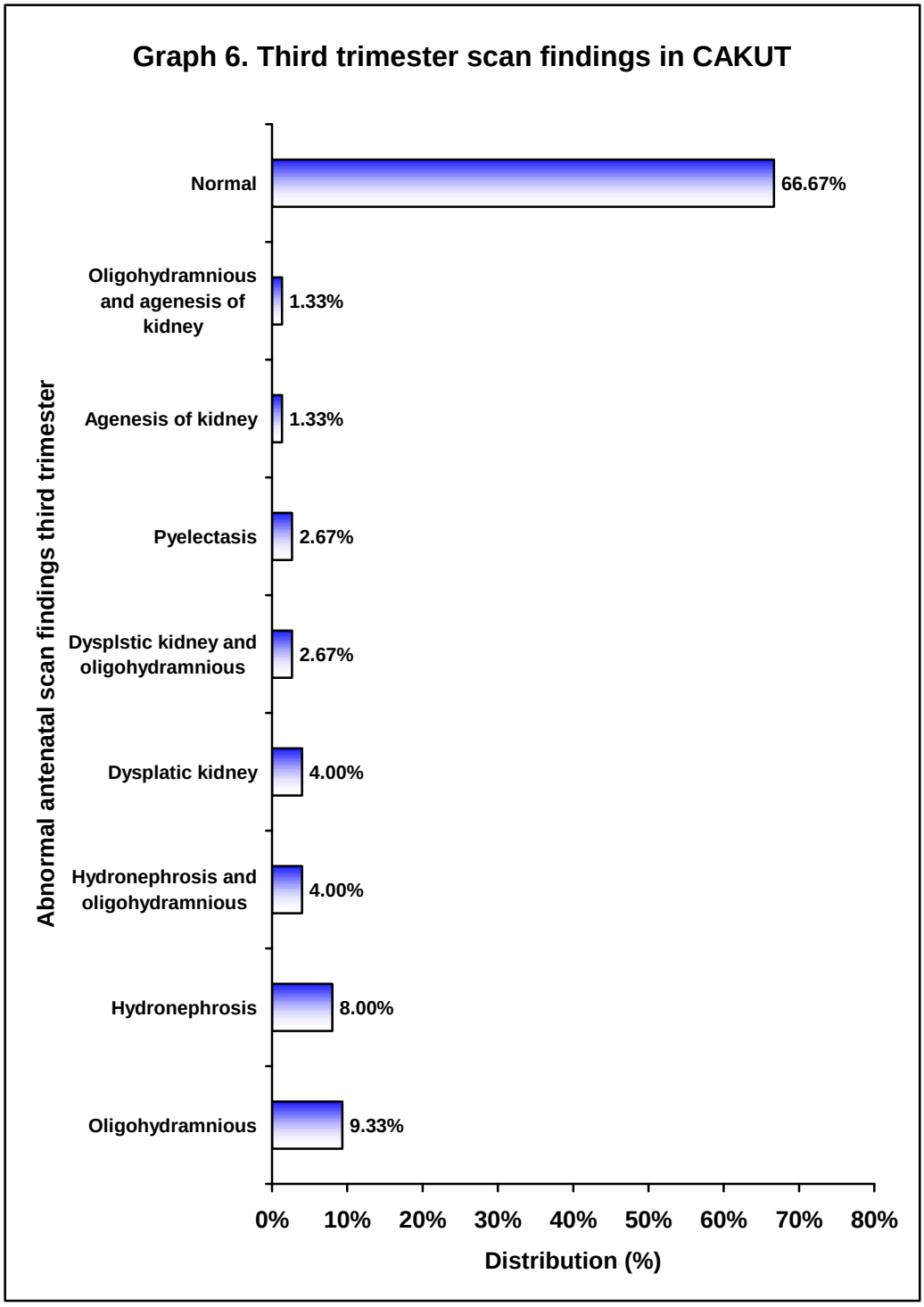
Abnormal Antenatal scan findings second trimester	Abnormal findings (n=75)	
	Number	**Percentage**
Agenesis of Kidney	1	1.33
Dysplastic kidney	1	1.33
Dysplastic kidney and oligohydramnios	1	1.33
Hydronephrosis	2	2.67
Normal	70	93.33
Total	**75**	**100.00**



In the present study none of the children had abnormal imaging findings during first trimester scan while, hydronephrosis was the common finding during second trimester scan noted in 2.67% of the children.

Table 6. Third trimester scan findings in children with CAKUT.

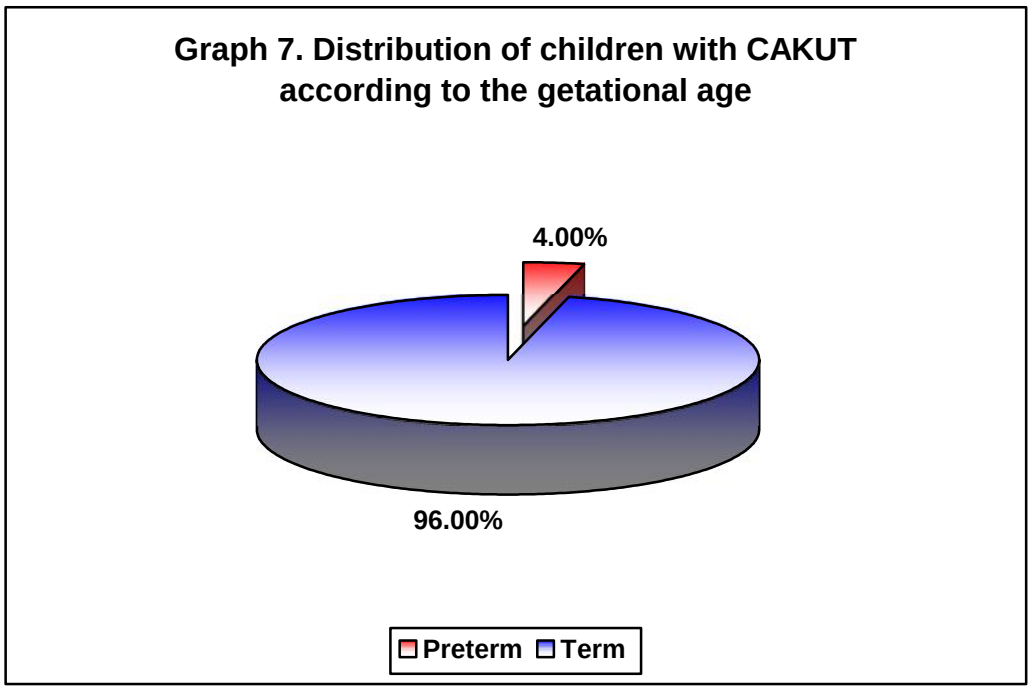
Abnormal Antenatal scan findings third trimester	Abnormal findings (n=75)	
	Number	**Percentage**
Oligohydramnios	7	9.33
Hydronephrosis	6	8.00
Hydronephrosis and oligohydramnios	3	4.00
Dysplastic kidney	3	4.00
Dysplastic kidney and oligohydramnios	2	2.67
Pyelectasis	2	2.67
Agenesis of Kidney	1	1.33
Oligohydramnios and agenesis of Kidney	1	1.33
Normal	50	66.67
Total	**75**	**100.00**



In this study during third trimester scan, Oligohydramnios was noted among 9.33% of the children.

Table 7. Distribution of children with CAKUT according to the gestational age

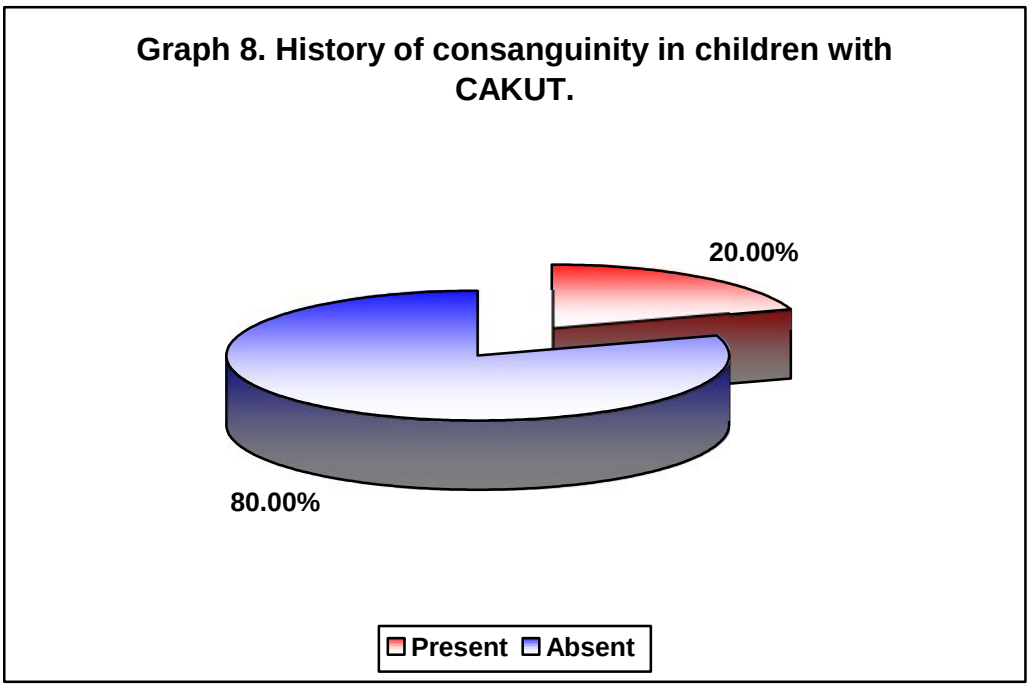
Gestational age	Abnormal findings (n=75)	
	Number	**Percentage**
Term	72	96.00
Preterm	3	4.00
Total	**75**	**100.00**



In the present study majority of the children term (96%) and 4% of the children were preterm.

Table 8. History of consanguinity in children with CAKUT

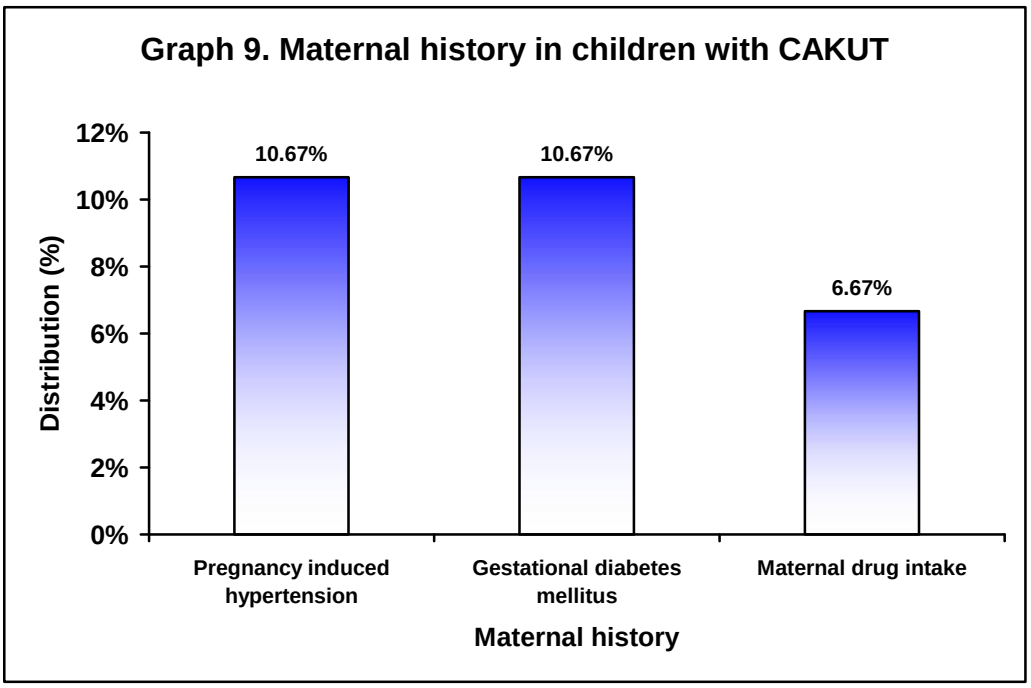
History of consanguinity	Abnormal findings (n=75)	
	Number	**Percentage**
Present	15	20.00
Absent	60	80.00
Total	**75**	**100.00**



In this studyhistory of consanguineous marriage was noted among 20% of the children with CAKUT.

Table 9. Maternal history in children with CAKUT.

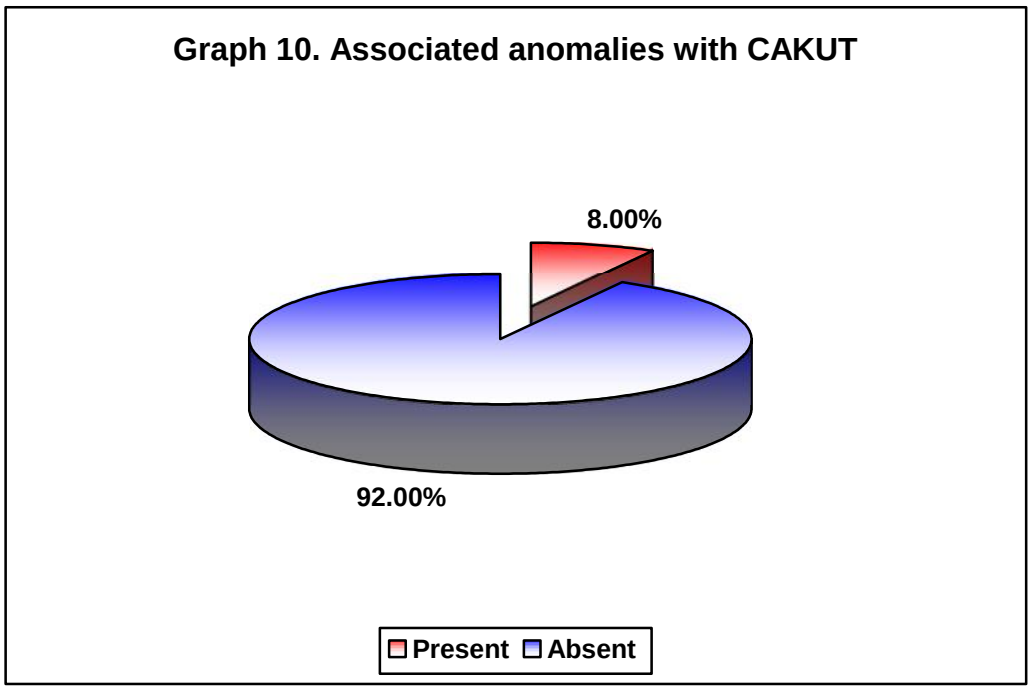
Maternal history	Abnormal findings (n=75)	
	Number	Percentage
Pregnancy induced hypertension (PIH)	8	10.67
Gestational diabetes mellitus (GDM)	8	10.67
Maternal drug intake	5	6.67



In the present study, most of the mother's having children with CAKUT had maternal history of pregnancy induced hypertension (PIH) and GDM (10.67% each).

Table 10. Associated anomalies with CAKUT.

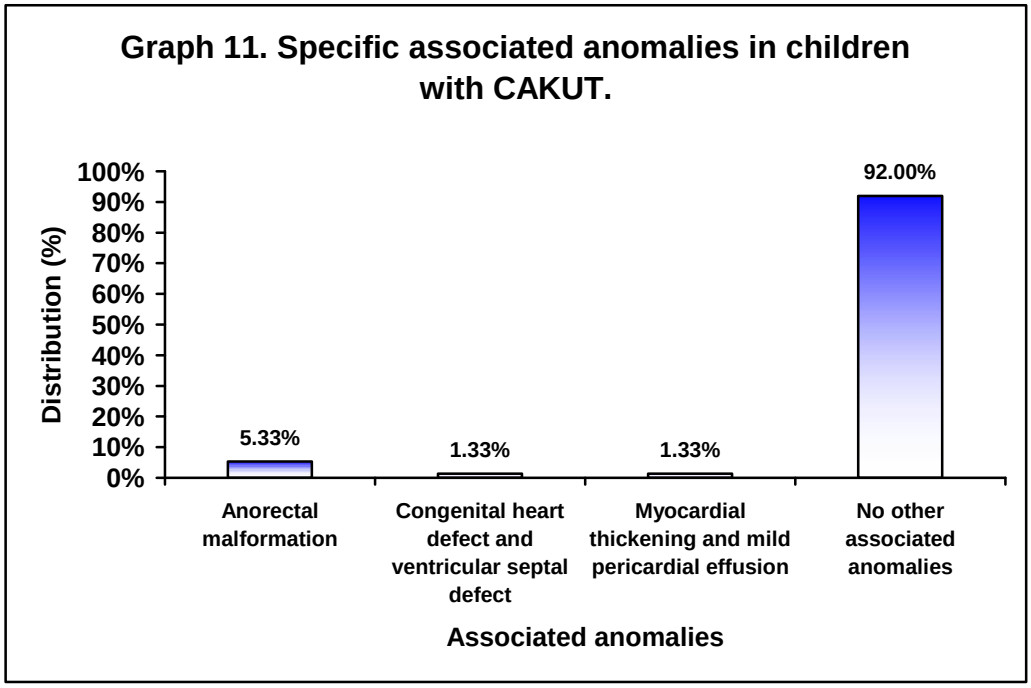
Other associated anomalies	Abnormal findings (n=75)	
	Number	**Percentage**
Present	6	8.00
Absent	69	92.00
Total	**75**	**100.00**



In this study 8% of the children had other associated anomalies.

Table 11. Specific associated anomalies in children with CAKUT.

Associated anomalies	Abnormal findings (n=75)	
	Number	**Percentage**
Anorectal malformation	4	5.33
Congenital heart defect and ventricular septal defect	1	1.33
Myocardial thickening and mild pericardial effusion	1	1.33
No other associated anomalies	69	92.00
Total	**75**	**100.00**

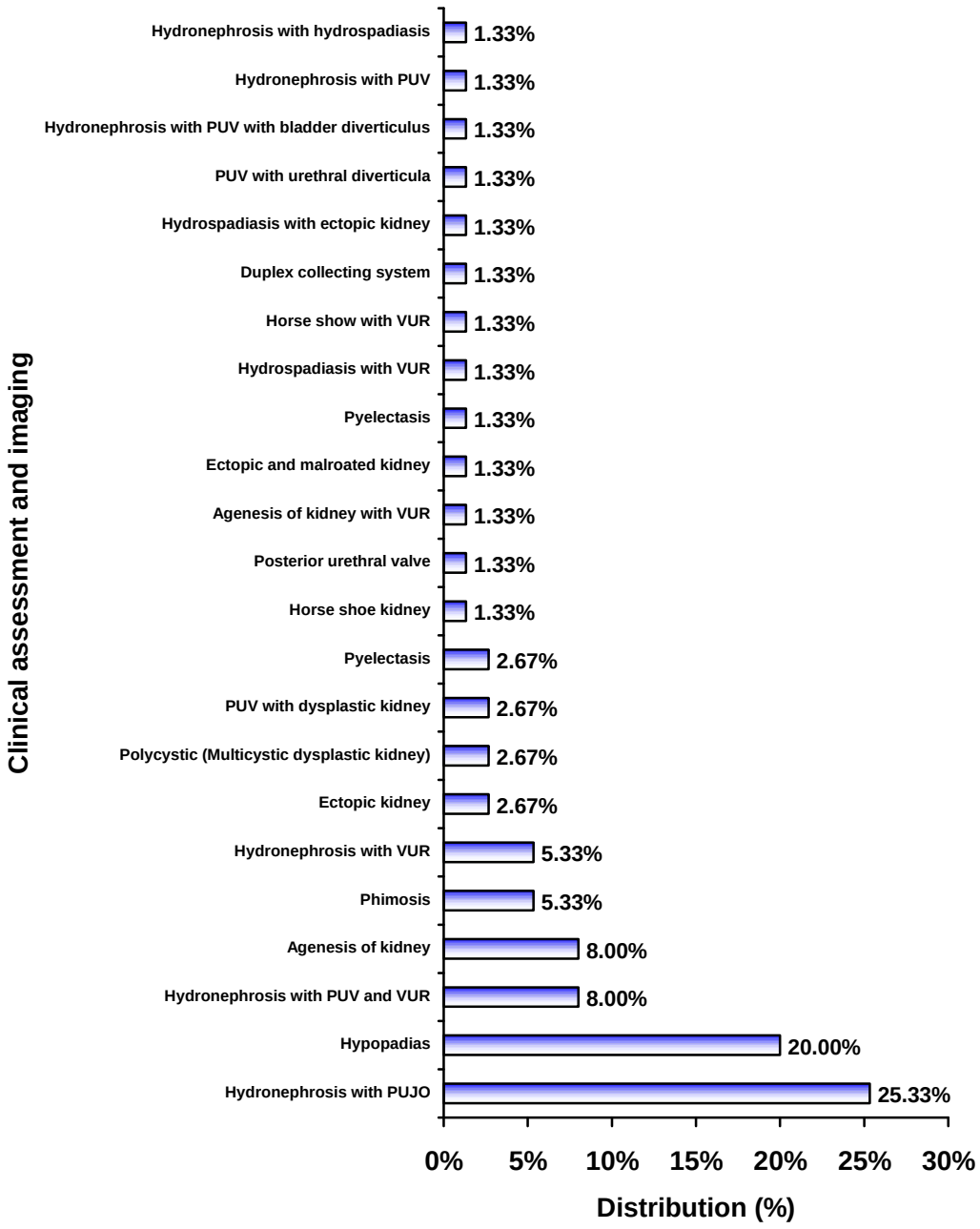


In the present study anorectal malformation was the common associated malformation noted among 5.33% of the children.

Table 12. Diagnosis of CAKUT based on clinical assessment and imaging

Diagnosis of CAKUT based on clinical assessment and imaging.	**Abnormal findings (n=75)**	
	Number	**Percentage**
Hydronephrosis with PUJO	19	25.33
Hypospadias	15	20.00
Hydronephrosis with PUV and VUR	6	8.00
Agenesis of kidney	6	8.00
Phimosis	4	5.33
Hydronephrosis with VUR	4	5.33
Ectopic kidney	2	2.67
Polycystic (Multicystic dysplastic kidney)	2	2.67
PUV with dysplastic kidney	2	2.67
Pyelectasis	3	4.00
Horse shoe kidney	1	1.33
Posterior urethral valve	1	1.33
Agenesis of kidney with VUR	1	1.33
Ectopic and malroated kidney	1	1.33
Hydrospadiasis with VUR	1	1.33
Horse show with VUR	1	1.33
Duplex collecting system	1	1.33
Hydrospadiasis with ectopic kidney	1	1.33
PUV with urethral diverticula	1	1.33
Hydronephrosis with PUV with bladder diverticulus	1	1.33
Hydronephrosis with PUV	1	1.33
Hydronephrosis with hydrospadiasis	1	1.33
Total	**75**	**100.00**

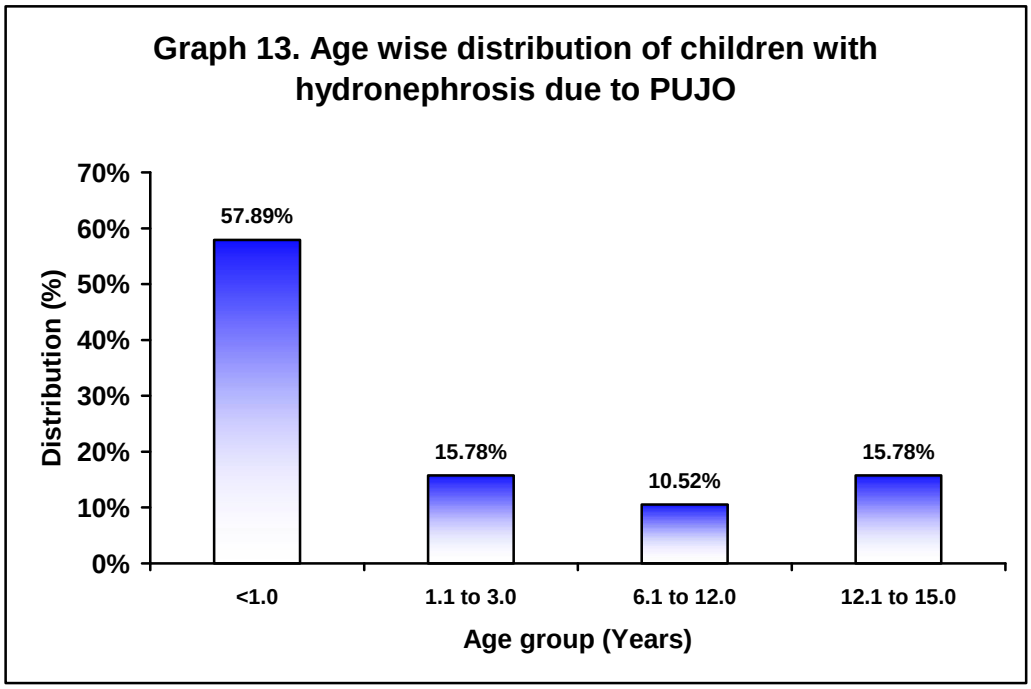
Graph 12. Distribution of children with CAKUT according to the various anomalies based on clinical assessment and imaging



In this study based on clinical assessment and imaging, Hydronephrosis with PUJO (25.33%) was the common CAKUT followed by Hypospadias (20%). The other CAKUT are as shown in table 12 and graph 12.

Table 13. Age wise distribution of children with hydronephrosis due to PUJO

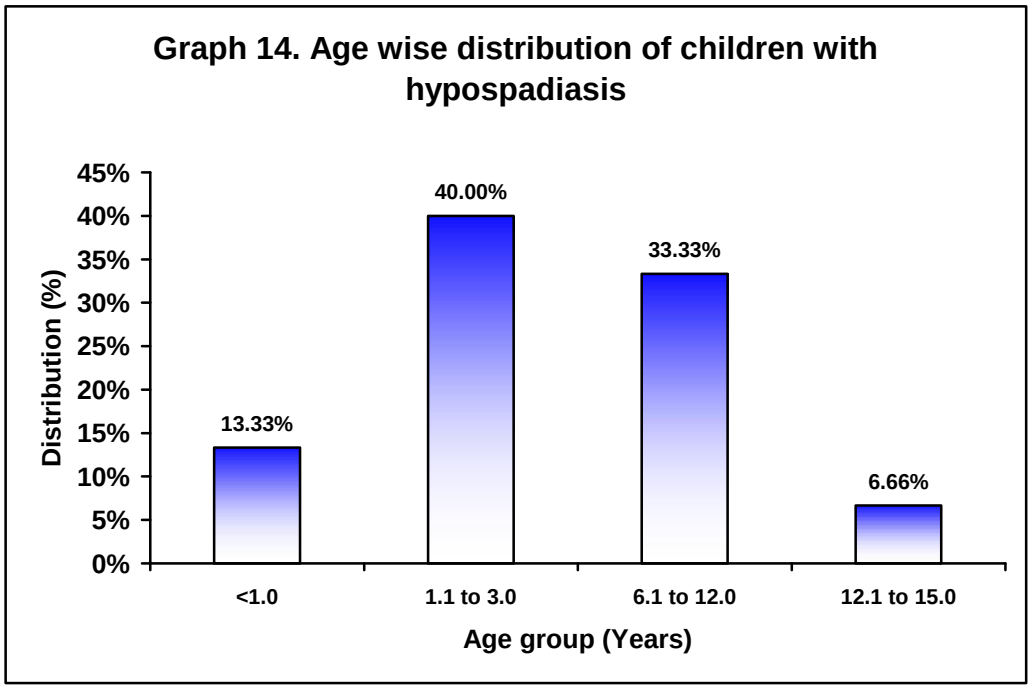
Age group	Distribution (n=19)	
	Number	**Percentage**
< 1	11	57.89
1.1 – 3	3	15.78
6.1 – 12	2	10.52
12.1 - 15	3	15.78
Total	**19**	**100.00**



In the present study most of the children with hydroneprhrosis with PUJO were aged < 1 year (57.89%).

Table 14. Age wise distribution of children with hypospadiasis

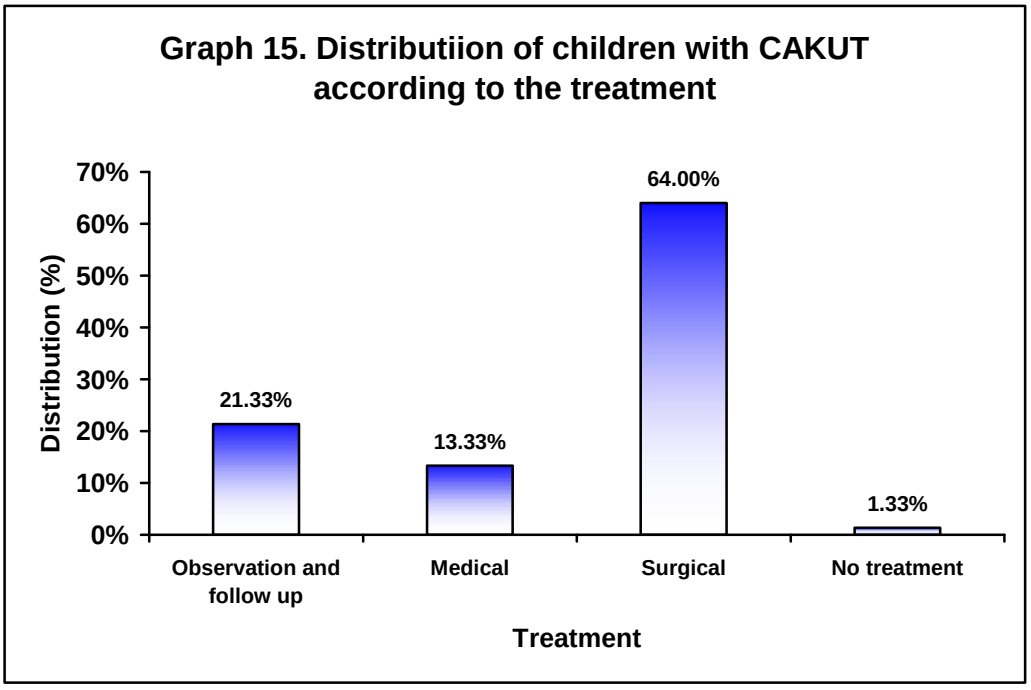
Age group	Distribution (n=19)	
	Number	**Percentage**
< 1year	2	13.33
1.1 – 3	6	40.00
3.1 – 6	5	33.33
6.1 – 12	1	6.66
Total	**19**	**100.00**



In the present study most of the children with hypospadiasis were aged between 1.1 to 3.0 years (40%).

Table 15. Distribution of children with CAKUT according to the treatment

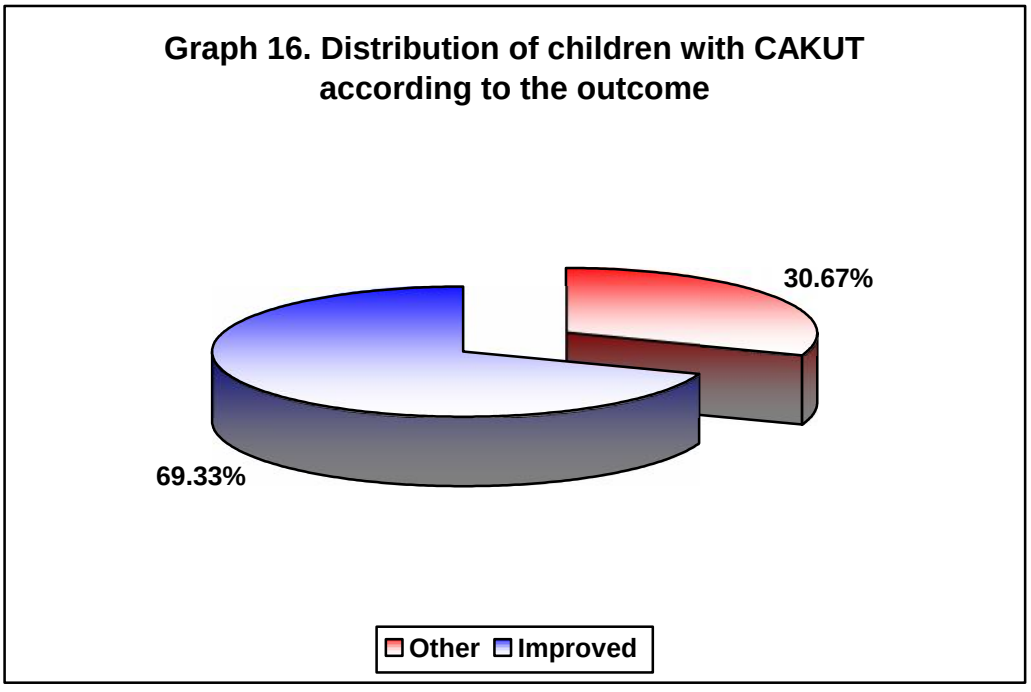
Treatment	Abnormal findings (n=75)	
	Number	**Percentage**
Observation and follow up	16	21.33
Medical	10	13.33
Surgical	48	64.00
No treatment	1	1.33
Total	**75**	**100.00**



In the present study majority of the children with CAKUT underwent surgical treatment.

Table 16. Distribution of children with CAKUT according to the outcome

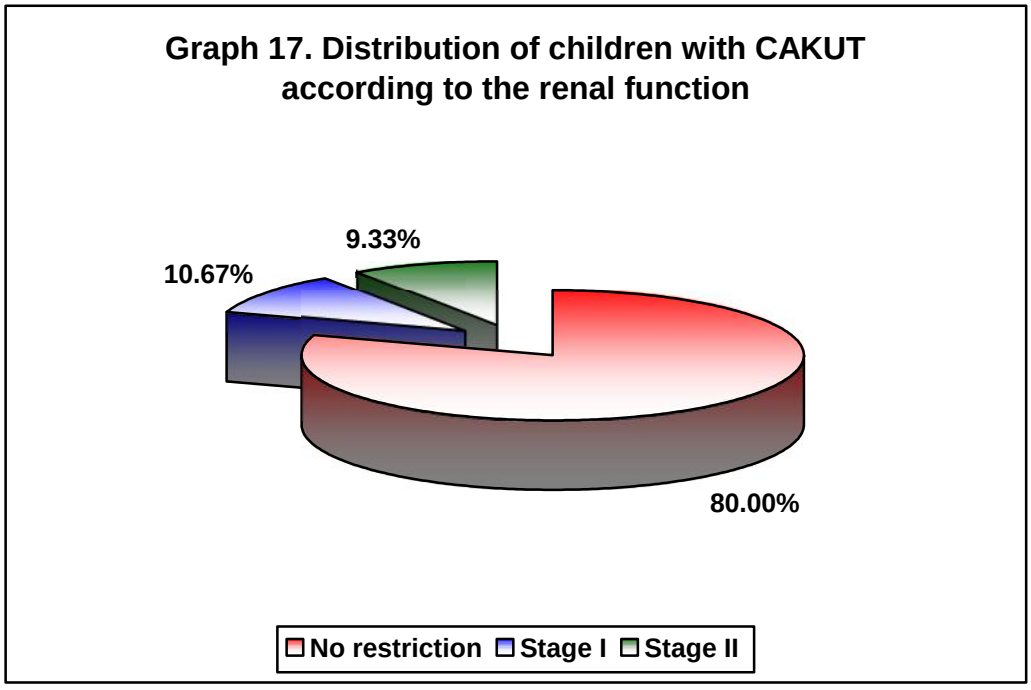
Outcome	Abnormal findings (n=75)	
	Number	**Percentage**
Improved	52	69.33
Other	23	30.67
Total	**75**	**100.00**



In this study majority of the children with CAKUT improved (69.33%).

Table 17. Children with CAKUT according to the renal function

Renal function based on AKIN criteria	Abnormal findings (n=75)	
	Number	**Percentage**
No restriction	60	80.00
Stage I	8	10.67
Stage II	7	9.33
Total	**75**	**100.00**



In the present study majority of the children with CAKUT had no restriction of renal function (80%) while, stage I renal function according to the AKIN criteria was noted in 10.67% of the children and stage II renal function was noted in 9.33% of the children.

DISCUSSION

Congenital anomalies of kidney and urinary tract occur frequently and incorporate a wide spectrum of anomalies which result due to developmental defects in urinary system. Prevalence of developmental defects of urinary system in isolation is established to be around 0.1% with antenatal ultrasonography and over 1% with postnatal ultrasonography. Chronic kidney disease found in 34-59% and 31% of chronic irreversible renal failure in children. They are cause of an immense distress to the family and impose a huge financial burden for treatment. As there are no studies done to estimate their prevalence, clinical course and outcome in the developing world,[48] hence, the present study was planned to determine the prevalence of CAKUT in children till 18 years of age and other associated anomalies and to assess the renal dysfunction according to AKIN's criteria.

This hospital based cross-sectional study for the period of one year from January 2018 to December 2018 was performed among the children admitted in the Department of Paediatrics, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. During the study period there were 7893 admission in the paediatric ward. Among them 81 cases were diagnosed to have CAKUT. Of them, 6 cases (0.07%) were previously diagnosed cases of CAKUT. Hence prevalence of CAKUT after excluding previously diagnosed cases was 0.95%. that is, 9.5 /1000 live births.

The prevalence of developmental defects of urinary systems noted in the present study was low compared to the study by Sallout B et al.[53] (2008) from Saudi Arabia which reported prevalence of as 21.28 per 1000 pregnancies antenatally and at birth the prevalence was 19.80 per 1000 live births which higher compared to the

present study. The study by Bondagji NS et al.[54] (2014) reported prevalence of CAKUT as 3.26 per 1000 births.

In contrast to the findings of the present study, Li ZY et al.[52] (2019) in Zhejiang Province, China. Studied the epidemiology of CAKUTs. 1.60 per 1000 livebirths was the prevalence of developmental defects of urinary systems. A 4 per 1000 newborn was the prevalence in murmansk county birth registry in Russia,[55] which was also very low compared to the present study. Data from two birth-defect registries from France[56] reported prevalence rate of CAKUTs as 4.0 per 10,000 which was lower than the present study.

Limited data is available regarding prevalence and pattern of urinary system anomalies in Indian children and there is a need to study the clinical course, their prevalence and population at risk, and outcome. However, a similar study by Prashar N. et al.[57] (2016) conducted in Jammu India reported 3.36 per 1000 births of urinary system anomalies at SMGS Hospital in one year, which was low compared to the present study.

The direct comparison of prevalence rates with the present study could not be done with above mentioned studies[52-57] due to methodological differences like varied sample size and also the different populations involved for example the prevalence rate calculated in the present study was from total number of children admitted during the study period and the children's age ranged between three days to 18 years while, the prevalence derived from the other studies is based on newborn/deliveries during study period. Also the prevalence of congenital anomalies vary from country to country due to the various racial, socio-cultural and ethnic differences.[58] In the Middle East, the marriage between blood relatives is common so prevalence there is 2-2.5%.

In England and US, the prevalence is 2% and 2-3% respectively.[59] Higher usage of alcohol, cigarettes and substance abuse in the Western population could explain this similarity in occurrence there where consanguinity was rare. The lower rate prevalence of CAKUT in the present study may be due to the fact that limited exposure of women in India to the teratogenic factors including abuse of substance, alcohol and smoking.[49]

In this study hypospadiasis was the common CAKUT diagnosis based on clinical assessment followed by Hydronephrosis. However, after the investigations, especially USG and clinical assessment, Hydronephrosis with PUJO (25.33%) was the common CAKUT followed by Hypospadias (20%). These observations were consistent with the studies by Prashar N. et al.[57] (2016) who reported that, of all urinary system anomalies, hydronephrosis was the most frequent abnormality seen. Similar observations were reported by Bondagji NS.[54] (2014) where 51.1% hydronephrosis cases were reported.

Majority of the children with CAKUT had no restriction of renal function (80%) while, stage I renal function according to the AKIN criteria was noted in 10.67% of the children and stage II renal function was noted in 9.33% of the children.

Male predominance is reported most commonly in several studies. 2:1–3:1 was the gender ratio.[54,60] In the present study majority of the frequency of CAKUT was very high among males compared to females (89.33% vs 10.67%) with male to female ratio of 8.3:1 suggesting male preponderance. Such observations were also documented by Prashar N. et al.[57] (2016) where majority of the affected fetuses were males, (63.46%) and (34.61%) were girls. The male preponderance noted in the present study was also comparable with a recent retrospective hospital-based study by

Li ZY et al.[52] (2019) from China where proportion of males and females with CAKUT was 56.73% and 39.28% respectively and the authors also reported the high risk in males.

More than half of the of the children (57.33%) aged less than or equal to one year were diagnosed to have CAKUT and the age among the children with CAKUT ranged between three days to 18 years. The mean age was in children with CAKUT was 2.83±4.11 years and the median age was 0.91 years. Hydronephrosis due to PUJO was commonly seen in children aged less than one year and hypospadiasis was common among the children aged between 1.1 to 3 years (33.3%). These observations could not be compared with other studies due to lack similar data in the literature as most of the studies have derived prevalence from either newborns from deliveries.

In the present study most of the cases with CAKUT belonged to socio economic class III (57.33%) according to the Modified B. G. Prasad's classification.[61] With regard to clinical characteristics, most of the cases presented with decreased stream of urine (26.66%) while 30.67% of the cases were asymptomatic. None of the children had abnormal imaging findings during first trimester scan while, among few of the children hydronephrosis was evident during second trimester scan (2.67%) and oligohydramnios was noted among 9.33% of the children during third trimester scan. In a study conducted Karambelkar GR et al.[48] (2016) in Pune, 35% of children with CAKUT demonstrated presence of congenital renal or urinary tract anomaly in postnatal scan which was very high compared to the present study.

With regard to birth history in our study, term delivery was noted in the majority of the children (96%) and 4% of the children were preterm. This observation was consistent with a study conducted by Karambelkar GR et al.[48] (2016) where

frequency of CAKUT was high in children with term gestation as compared to preterm gestation. Another study by Bondagji NS.[54] (2014) reported the occurrence more in term delivery a finding strongly in agreement with the present study. On the other hand a study by Tain et al.[2] (2016) found out that, baby born preterm has more incidence rate.

In this study some of the children with CAKUT had history of consanguineous marriage (20%). In a study by Prashar N et al.[57] (2016), 3.84% anomalous fetuses were out of consanguineous marriages while in study by Bondagji NS[54] (2014), [8] 40.4% were from the same. Such a high incidence of anomalies out of consanguineous marriage in study by Bondagji NS[54] (2014) may be because of high rate of these marriages in Saudi Arabia as reported by author only.

In the present study, PIH and GDM were the common maternal risk factors among the children with CAKUT (10.67% each) and history of maternal drug intake was noted in 6.67% of the women. Similarly a study by Tain et al.[2] (2016) also reported that, gestational diabetes, pregestational diabetes carries more risk for the incidence of anomalies. On the contrary a study by Karambelkar GR et al.[48] (2016) reported no history of maternal risk factors in children with CAKUT.

In this study other associated anomalies were noted in 8% of the children with CAKUT and anorectal malformation was the common associated malformation noted among 5.33% of the children. The rate of other anomalies noted in the present study was low Compared to a study by Prashar N et al.[57] (2016) who reported that, out of 52 cases diagnosed with urinary system anomalies, 11 (21.15%) had other associated anomalies including central nervous system, cardiovascular, musculoskeletal, gastrointestinal systems and 41 cases (78.85%) had isolated urinary system anomalies.

A study conducted by Karambelkar GR et al.[48] (2016) Other associated congenital anomalies were present in 17.5% subjects which slightly high compared to the present study. In another study conducted by Li ZY et al.[52] (2019) 22.69% of the children with CAKUT had associated malformations and congenital heart defects were the most common anomalies. In another study by Bondogji NS,[54] (2014) associated anomalies were present in 26.2% of the children with CAKUT.

In the present study majority of the children with CAKUT underwent surgical treatment (64%) and majority of the children with CAKUT improved (69.33%). These observations were partly in agreement with a study by Bondogji NS.[54] who reported that 17 cases showed improvement without surgery in moderate and severe hydronephrosis, and 10 cases needed surgery.

Strengths and limitation of the study

Overall the present study showed prevalence of CAKUT in children till 18 years of age as 9.5 per 1000. The strength of the study was that prevalence of CAKUT was derived from the wide age range (from newborn to 18 years). Although it reflects the true prevalence rate but we presume that the prevalence rate derived in the present study is underestimated as the present study was limited to the children who were admitted to our tertiary care centre and many children remain asymptomatic and remain undiagnosed till the adult age and such children were not accounted, which was the limitation of the study. The other limitations of the study were, this was a hospital based study involving relatively a smaller sample which limits the generalizability of the findings to the entire population. The data from this study can act as a base for further etiologic research so that efficient measures can be taken to

prevent the CAKUT. Burden of the disease and mortality of associated with the diseases can be decreased by early recognition.

Recommendations

In view of the potential limitation of the present study, large multicentric population based studies are required to explore the real epidemiology of CAKUT in the study area.

CONCLUSION

Based on the observations from this study it may be concluded that, prevalence of CAKUT in children till 18 years of age is 0.95% that is, 9.5 per 1000 children. Hydronephrosis is the common anomaly due to pelvic ureteric junction obstruction followed by hypospadias which can be easily recognized and managed appropriatly.

SUMMARY

Developmental defects of urinary systems occur more commonly and consists of a large spectrum of malformations which result due to developmental malformations in urinary system. The present study was undertaken to describe the epidemiology of CAKUT in children till 18 years of age and other associated anomalies and to assess the renal dysfunction according to AKIN's criteria.

This hospital based cross-sectional study for the period of one year from January 2018 to December 2018 was carried out among the children admitted under the Department of Paediatrics, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. The important findings of the study are highlighted as below.

- During the study period there were 7893 admission in the paediatric ward. Among them 81 cases were diagnosed to have CAKUT. Of them, 6 cases (0.07%) were previously diagnosed cases of CAKUT.
- The prevalence of CAKUT was 0.95% or 9.5 per 1000.
- Hypospadiasis was the common CAKUT diagnosis based on clinical assessment followed by Hydronephrosis.
- Based on clinical assessment and imaging, Hydronephrosis with PUJO (25.33%) was the common CAKUT followed by Hypospadias (20%).
- Other associated anomalies were noted in 8% of the children and anorectal malformation was the common associated malformation noted among 5.33% of the children.

- Majority of the children with CAKUT had no restriction of renal function (80%) while, stage I renal function according to the AKIN criteria was noted in 10.67% of the children and stage II renal function was noted in 9.33% of the children.

- Majority of the children with CAKUT underwent surgical treatment (64%) and improved (69.33%).

- Majority of the cases with CAKUT were diagnosed among males (89.33%) while 10.67% of the cases were diagnosed among females.

- Most of the CAKUT cases were aged less than or equal to one year (57.33%). The mean age was 2.83±4.11 and the median age was 0.91 years.

- Hydronephrosis due to PUJO was commonly seen in children aged less than one year and hypospadiasis was common among the children aged between 1.1 to 3 years (40%)

- Most of the cases with CAKUT belonged to socio economic class III (57.33%) according to the Modified B. G. Prasad's classification.

- Most of the cases presented with decreased stream of urine (26.66%) while 30.67% of the cases were asymptomatic.

- None of the children had abnormal imaging finding during first trimester scan while, hydronephrosis was the common finding during second trimester scan noted in 2.67% of the children. During third trimester scan, Oligohydramnios was noted among 9.33% of the children.

- Majority of the children term (96%) and 4% of the children were preterm.

- History of consanguineous marriage was noted among 20% of the children with CAKUT. Most of the mother's having children with CAKUT had maternal history of PIH and GDM (10.67% each).

Overall, the prevalence of CAKUT in children till 18 years of age is 0.95% that is, 9.5 per 1000 children. Hydronephrosis is the common anomaly followed by pelvic ureteric junction obstruction and hypospadias which are easily treatable.

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ANNEXURE I – CONSENT FORM

CONSENT FOR PARTICIPATION IN RESEARCH

"ASSIGNING CAUSE OF STILLBIRTH - COMPARISON OF TWO METHODS: ONE YEAR HOSPITAL BASED OBSERVATIONAL STUDY"

Principal Investigator: Dr._____

Guide: Dr. _____

Co-Guide: Dr._____

You are hereby requested to involve yourself and your child in the above said research to be conducted at KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2018 to December 2018 by me.

Introduction

Congenital anomalies of kidney and urinary tract (CAKUT) are group of urinary systems anomalies. In spite of the abnormality may not become evident until later in life, it is present since birth. They results from deviation of the normal development of the urinary system. Ureter, urinary bladder and urethra are the other parts of the urinary tract that may be affected. They represents 20% to 50% of all fetal congenital anomalies, and 30% to 60% of cases may lead to childhood-onset chronic kidney disease (CKD), often they are asymptomatic. One of the major causes of renal replacement therapy (RRT) and premature mortality in the young adult population

Voluntary participation

You and your child's participation in this study is your voluntary decision. Whether to participate or not to participate will not affect your current or future relationship with the KLES Dr. Prabhakar Kore Hospital and Medical Research

Centre, Belgaum. You are free to discontinue the participation in the study at any time for any reasons and you will not be paid any reimbursement for participation in the research**.**

Risk and benefits

There are no potential risks and discomforts associated with any procedure involved in our study. The benefits of taking part in this research is your valuable contribution to medical research.

Withdrawal from the study

You can withdraw at any time from the study. There will be no penalty for withdrawal.

Privacy and Confidentiality

The only people who will know that you are a research participant are member of the research team. No information provided by you, during research will be disclosed to others without your written consent. When the results of the research are published or discussed in the conferences, no information will be disclosed that would reveal your identity. Any information obtained in connections with this study and that can be identified with you remain confidential and will be disclosed only with your permission.

Queries

If you have any queries you may contact

Dr. _____

Post Graduate Student

Department of Pediatrics

JNMC, Belagavi-590010

Phone No._____

Dr. _____

MD (Pediatrics),

Professor & HOD, Department of Pediatrics

I/C Division of pediatric nephrology

JNMC, Belagavi-590010

Dr. _____

MS, MCH

Professor & HOD

Department of pediatric Surgery

JNMC, Belagavi-590010

If you have any questions about your rights or research participation you may contact

Dr. Roopa Bellad

Chairperson, Ethical Committee

JNMC Belagavi-590010

Phone No.9480275601

You will be given a copy of this form for your information and to keep for your record

STATEMENT OF CONSENT

I hereby voluntarily agree for my and my child's participation in this study. I understand that even if I choose to allow my child to take part in this study I have the liberty to withdraw at any time. My signature below indicates that I have read or have been told about this entire consent form including the risks and benefits and have had all my questions answered. I will be given a copy of this consent form.

Signature of the authorized representative/ parent: _____

Date: _____

Name: _____

Relation to the Subject: _____

Signature of the witness: _____

Date: _____

Name: _____

Signature of investigator: _____

Date: _____

Name: _____

ANNEXURE I – ETHICAL CLEARANCE LETTER

K.L.E.UNIVERSITY'S
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
(Accredited 'A' Grade by NAAC)

Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu

Phone: (+ 91-(0)831 Office : 2471350
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 73 **Date: 22/11/2017**

To,

PG student in Paediatrics,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"PREVALENCE OF CONGENITAL ANOMALIES OF KIDNEY AND URINARY TRACT (CAKUT) IN CHILDREN: A ONE YEAR CROSS SECTIONAL STUDY AT KLE'S DR PRABHAKAR KORE HOSPITAL & MRC BELAGAVI"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

ANNEXURE II – PROFORMA

ANNEXURE III – PROFORMA

Title: Prevalence of Congenital Anomalies of Kidney and Urinary Tract (CAKUT) in Children: A One Year Cross Sectional Study at KLE'S Dr. Prabhakar Kore Hospital & MRC Belagavi

Principal Investigator:
Dr. _____
Post Graduate Student
Department of Pediatrics
JNMC, Belgaum.

Guide: DR. _____

Co Guide: DR. _____

Serial No: IP No:

Name: Age: Sex:

Address: Contact No:

DOA:

Socioeconomic status:

Chief Complaints:

History In Brief:

Past history

H/O Diabetes

H/O Hypertension

H/O Hypothyroidism

H/O Siblings with similar complaints

H/O Drug intake

Antenatal history

1ST TRIMESTER SCAN:

2ND TRIMESTER SCAN:

Amniotic fluid index-

Any other anomalies-

3RD TRIMESTER SCAN:

Amniotic fluid index-

Any other anomalies-

H/O Consanguinity

H/O Maternal

Ureter duplex-

Renal agenesis-

H/O Hypertension/PIH

H/O Drug intake

Angiotensin-converting enzyme (ACE) Inhibitors-

Angiotensin Receptor Blockers-

H/O infection

H/O Radiation

Natal history (Birth history):

Gestational Age at Birth-

Birth weight-

Type of Delivery-LSCS/NORMAL

If LSCS Indication-

Place of Delivery-

Cried at-

APGAR Score-

Admission to NICU (with reason)-

Postnatal history:

HIE-

Jaundice-

Feeding difficulty-

RDS-

Infections-

Developmental history:

Gross motor Fine motor Social & Adaptive

Language

Examination:

Vitals:

Heart rate (/Minute):

Respiratory rate (/Minute):

Temperature (^{0}C):

Blood pressure (mm Hg):

Anthropometry:

Parameter	**Child's measurement**	**Expected**	**Percentile**
Weight (Kg)			
Height/Length (cm)			
HC (cm)			
CC			
MAC			

General physical examination:

Pallor:	Bleeding:
Clubbing:	dehydration:
Cyanosis:	JVP:
Lymphadenopathy:	CFT:
Oedema:	Rashes:
Icterus:	Grunting:
Retractions:	Shock:
Congenital markers:	Acidosis:

Systemic examination:

Cardiovascular system:

Respiratory system:

Per abdomen/renal system:

Central nervous system:

Genitals:

Undescended testis-

Hydrocele-

Vericocele-

Hypospadias-

Epispadias-

Stretched penile length

Probable Diagnosis:

Investigations

Complete blood count

HB	TC	Neutrophil	Lymphocyte	Eosinophil	RBC	Platelet count	ESR	Retic count

Calcium	Phosphate	Magnesium	Albumin	Alk phosphtase	Intact PTH	Uric acid

Electrolytes

Sodium	Potassium	Chlorides	Bicarbonate

Arterial blood gas:

PH	PCO2	PO2	SO2	BE	HCO3

Acute Kidney Injury (AKIN'S Criteria) (In pts presenting with AKI)

AKI	At presentation	On follow up
Creatinine		
BUN		

Urine Examination (Routine & Microscopy)

Color: Nitrite:

Appearance: Bilirubin:

PH: Urobilinogen:

Specific Gravity: RBC:

Protein: WBC:

Glucose: Epithelial cells:

Ketone bodies: Casts:

Bacteria:

Urine Protiene/Creatinine Ratio:

Urine Culture & sensitivity:

Radiological:

1. *USG abdomen*

SL NO	IP NO	

2. MCU

SL NO	IP NO		
		Bladder capacity	
		Cantour*Regular/Irregular	
		VU reflex	

3. MR urography

SL NO	IP NO	

4. Nuclear scan (DTPA/DMSA)

SL NO	IP NO	

5. IVP

SL NO	IP NO	

Final Diagnosis:

Treatment:

Discharged	**Operation/Procedure**	**AKI** **eGFR(ml/min/1.73m2)**
DOD:		
TOD:		

Final Outcome:

ANNEXURE IV – KEY TO MASTER CHART

AgK	-	Agenesis of Kidney
AM	-	Anorectal malformation
B	-	Observation
CHD,VSD	-	Congenital heart defect and ventricular septal defect
Cms	-	Centimeters
d	-	Days
DK	-	Dysplastic kidney
DK,Olig	-	Dysplastic kidney and oligohydramnious
f	-	Female
gm	-	Gram
HDN	-	Hydronephrosis
HDN,Olig	-	Hydronephrosis and oligohydramnious
I	-	Improved
IVP	-	Intravenous pyelogram
Kg	-	Kilogram
m	-	Male
M	-	Medical
MAC	-	Mid arm circumference
MCU	-	Micturating cycstoureterography
mg/dL	-	Milligrams per deciliter
mm Hg	-	Millimeters of mercury
mo	-	Months
MT,MPE	-	Myocardial thickening and mild pericardial effusion

N	-	No
No.	-	Number
O	-	Others
Olig	-	Oligohydramnious
Olig,AgK	-	Oligohydramnious and agenesis of Kidney
P	-	Preterm
PLS	-	Pyelectasis
S	-	Surgical
T	-	Term
U	-	Follow up
y	-	Years
Y	-	Yes

Chief complaints

1	-	Decreased stream of urine and decreased urine output
2	-	Pain abdomen, fever, vomiting, burning micturition
3	-	Passing urine underneath of penis
4	-	Antenatal scan

Diagnosis

1	-	Agenesis of kidney
2	-	Horse shoe kidney
3	-	Ectopic kidney
4	-	Hydronephrosis with PUJO
5	-	Posterior urethral valve
6	-	Polycystic (Multicystic dysplastic kidney)
7	-	Hypospadias
8	-	Phimosis
9	-	Hydronephrosis with VUR
10	-	Hydronephrosis with PUJO with agenesis with ectopic kidney
11	-	Pyelectasis
12	-	Aqueous of kidney with VUR
13	-	Duplex collecting systems
14	-	UTI
15	-	Pyelectasis

Final diagnosis

1	-	Agenesis of kidney
2	-	Horse shoe kidney
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4	-	Hydronephrosis with PUJO
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11	-	Hydronephrosis with PUV and VUR
12	-	PUV with dysplastic kidney
13	-	Agenesis of kidney with VUR
14	-	Ectopic and malrotated kidney
15	-	Pyelectasis
16	-	Hypospadiasis with VUR
17	-	Pyelectasis
18	-	Horse show with VUR
19	-	Duplex collecting system
20	-	Hypospadiasis with ectopic kidney
21	-	PUV with urethral diverticula
22	-	Hydronephrosis with PUV with bladder diverticulum
23	-	Hydronephrosis with PUV
24	-	Hydronephrosis with hypospadiasis

ANNEXURE I – CONSENT FORM

CONSENT FOR PARTICIPATION IN RESEARCH

"ASSIGNING CAUSE OF STILLBIRTH - COMPARISON OF TWO METHODS: ONE YEAR HOSPITAL BASED OBSERVATIONAL STUDY"

Principal Investigator: Dr.Manoj Kaddlimatti

Guide: Dr. Mahantesh V Patil

Co-Guide: Dr. Santosh B Kurbet

You are hereby requested to involve yourself and your child in the above said research to be conducted at KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2018 to December 2018 by me.

Introduction

Congenital anomalies of kidney and urinary tract (CAKUT) are group of urinary systems anomalies. In spite of the abnormality may not become evident until later in life, it is present since birth. They results from deviation of the normal development of the urinary system. Ureter, urinary bladder and urethra are the other parts of the urinary tract that may be affected. They represents 20% to 50% of all fetal congenital anomalies, and 30% to 60% of cases may lead to childhood-onset chronic kidney disease (CKD), often they are asymptomatic. One of the major causes of renal replacement therapy (RRT) and premature mortality in the young adult population

Voluntary participation

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Risk and benefits

There are no potential risks and discomforts associated with any procedure involved in our study. The benefits of taking part in this research is your valuable contribution to medical research.

Withdrawal from the study

You can withdraw at any time from the study. There will be no penalty for withdrawal.

Privacy and Confidentiality

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Queries

If you have any queries you may contact

Dr. Manoj Kadlimatti,

Post Graduate Student

Department of Pediatrics

JNMC, Belagavi-590010

Phone No. 7829778527

Dr. Mahantesh V Patil

MD (Pediatrics),

Professor & HOD, Department of Pediatrics

I/C Division of pediatric nephrology

JNMC, Belagavi-590010

Phone No. 0831-2473777 Ext no. 1882, 4032.

Dr. Santosh B Kurbet

MS, MCH

Professor & HOD

Department of pediatric Surgery

JNMC, Belagavi-590010

Phone No. 0831-2473777 Ext no. 1386

If you have any questions about your rights or research participation you may contact

Dr. Roopa Bellad

Chairperson, Ethical Committee

JNMC Belagavi-590010

Phone No.9480275601

You will be given a copy of this form for your information and to keep for your record

STATEMENT OF CONSENT

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Signature of the authorized representative/ parent: _____

Date: _____

Name: _____

Relation to the Subject: _____

Signature of the witness: _____

Date: _____

Name: _____

Signature of investigator: _____

Date: _____

Name: _____

ANNEXURE I – ETHICAL CLEARANCE LETTER

K.L.E.UNIVERSITY'S
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
(Accredited 'A' Grade by NAAC)

Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu

Phone: (+ 91-(0)831 Office : 2471350
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 73 **Date: 22/11/2017**

To,

Dr. Manoj Kadlimatti,
PG student in Paediatries,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"PREVALENCE OF CONGENITAL ANOMALIES OF KIDNEY AND URINARY TRACT (CAKUT) IN CHILDREN: A ONE YEAR CROSS SECTIONAL STUDY AT KLE'S DR PRABHAKAR KORE HOSPITAL & MRC BELAGAVI"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

ANNEXURE II – PROFORMA

ANNEXURE III – PROFORMA

Title: Prevalence of Congenital Anomalies of Kidney and Urinary Tract (CAKUT) in Children: A One Year Cross Sectional Study at KLE'S Dr. Prabhakar Kore Hospital & MRC Belagavi

Principal Investigator:
Dr. MANOJ KADLIMATTI
Post Graduate Student
Department of Pediatrics
JNMC, Belgaum.

Guide: DR. MAHANTESH. V. PATIL

Co Guide: DR. SANTOSH. B. KURBET

Serial No: IP No:

Name: Age: Sex:

Address: Contact No:

DOA:

Socioeconomic status:

Chief Complaints:

History In Brief:

Past history

H/O Diabetes

H/O Hypertension

H/O Hypothyroidism

H/O Siblings with similar complaints

H/O Drug intake

Antenatal history

1ST TRIMESTER SCAN:

2ND TRIMESTER SCAN:

Amniotic fluid index-

Any other anomalies-

3RD TRIMESTER SCAN:

Amniotic fluid index-

Any other anomalies-

H/O Consanguinity

H/O Maternal

Ureter duplex-

Renal agenesis-

H/O Hypertension/PIH

H/O Drug intake

Angiotensin-converting enzyme (ACE) Inhibitors-

Angiotensin Receptor Blockers-

H/O infection

H/O Radiation

Natal history (Birth history):

Gestational Age at Birth-

Birth weight-

Type of Delivery-LSCS/NORMAL

If LSCS Indication-

Place of Delivery-

Cried at-

APGAR Score-

Admission to NICU (with reason)-

Postnatal history:

HIE-

Jaundice-

Feeding difficulty-

RDS-

Infections-

Developmental history:

Gross motor Fine motor Social & Adaptive

Language

Examination:

Vitals:

Heart rate (/Minute):

Respiratory rate (/Minute):

Temperature (^{0}C):

Blood pressure (mm Hg):

Anthropometry:

Parameter	**Child's measurement**	**Expected**	**Percentile**
Weight (Kg)			
Height/Length (cm)			
HC (cm)			
CC			
MAC			

General physical examination:

Pallor: Bleeding:

Clubbing: dehydration:

Cyanosis: JVP:

Lymphadenopathy: CFT:

Oedema: Rashes:

Icterus: Grunting:

Retractions: Shock:

Congenital markers: Acidosis:

Systemic examination:

Cardiovascular system:

Respiratory system:

Per abdomen/renal system:

Central nervous system:

Genitals:

Undescended testis-

Hydrocele-

Vericocele-

Hypospadias-

Epispadias-

Stretched penile length

Probable Diagnosis:

Investigations

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HB	TC	Neutrophil	Lymphocyte	Eosinophil	RBC	Platelet count	ESR	Retic count

Calcium	Phosphate	Magnesium	Albumin	Alk phosphtase	Intact PTH	Uric acid

Electrolytes

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Arterial blood gas:

PH	PCO2	PO2	SO2	BE	HCO3

Acute Kidney Injury (AKIN'S Criteria) (In pts presenting with AKI)

AKI	At presentation	On follow up
Creatinine		
BUN		

Urine Examination (Routine & Microscopy)

Color: Nitrite:

Appearance: Bilirubin:

PH: Urobilinogen:

Specific Gravity: RBC:

Protein: WBC:

Glucose: Epithelial cells:

Ketone bodies: Casts:

Bacteria:

Urine Protiene/Creatinine Ratio:

Urine Culture & sensitivity:

Radiological:

1. *USG abdomen*

SL NO	IP NO	

2. MCU

SL NO	IP NO		
		Bladder capacity	
		Cantour*Regular/Irregular	
		VU reflex	

3. MR urography

SL NO	IP NO	

4. Nuclear scan (DTPA/DMSA)

SL NO	IP NO	

5. IVP

SL NO	IP NO	

Final Diagnosis:

Treatment:

Discharged	**Operation/Procedure**	**AKI** **eGFR(ml/min/1.73m2)**
DOD:		
TOD:		

Final Outcome:

ANNEXURE IV – KEY TO MASTER CHART

AgK	-	Agenesis of Kidney
AM	-	Anorectal malformation
B	-	Observation
CHD,VSD	-	Congenital heart defect and ventricular septal defect
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Kg	-	Kilogram
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MAC	-	Mid arm circumference
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mg/dL	-	Milligrams per deciliter
mm Hg	-	Millimeters of mercury
mo	-	Months
MT,MPE	-	Myocardial thickening and mild pericardial effusion

N	-	No
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O	-	Others
Olig	-	Oligohydramnious
Olig,AgK	-	Oligohydramnious and agenesis of Kidney
P	-	Preterm
PLS	-	Pyelectasis
S	-	Surgical
T	-	Term
U	-	Follow up
y	-	Years
Y	-	Yes

Chief complaints

1	-	Decreased stream of urine and decreased urine output
2	-	Pain abdomen, fever, vomiting, burning micturition
3	-	Passing urine underneath of penis
4	-	Antenatal scan

Diagnosis

1	-	Agenesis of kidney
2	-	Horse shoe kidney
3	-	Ectopic kidney
4	-	Hydronephrosis with PUJO
5	-	Posterior urethral valve
6	-	Polycystic (Multicystic dysplastic kidney)
7	-	Hypospadias
8	-	Phimosis
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10	-	Hydronephrosis with PUJO with agenesis with ectopic kidney
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14	-	UTI
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Final diagnosis

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2	-	Horse shoe kidney
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21	-	PUV with urethral diverticula
22	-	Hydronephrosis with PUV with bladder diverticulum
23	-	Hydronephrosis with PUV
24	-	Hydronephrosis with hypospadiasis

Serial Number	In patient No.	Age	Age (years)	Age group	gender	Socio economic status	Chief complaints	Past history		History of similar complaints in sibl	Treatment history	Antenatal history					History of Consanguinity	Pregancy induced hypertension	Maternal dabetes	Maternal drug intake									Diagnosis	Investigations										Treatment	Outcome	AKIN's Criteria
								Diabetes mellitus	Hypertension			First trimester scan	Second trimester scan	Third trimester scan	gestational age	Other anomalies					SBP (mm Hg)	DBP (mm Hg)	Weight (Kg)	Height (cms)	Head circumference (cms)	MAC	Cardiovascular system	Respiratory system	Per abdomen	Central nervous system		Haemoglobin (gm%)	Bicarboanate	Serum Creatinine (mg/dL)	Serum Creatinine (mg/dL)@48hrs	Blood urea nitrogen (mg/dL)	Ratio	USG abdomen	MCU	MR urography	Nuclear scan	IVP	Final diagnosis			
1	891451	8d	0.021	1 or less	f	I	-	N	N	N	N	N	N	HDN,Olig	T	N	N	N	N	N	64	32	2.1	40	31	-	Nr	Nr	Nr	Nr	4	15.8	10	0.8	0.78	26	-	Y	N	N	N	N	4	M	I	-
2	884947	4mo	0.330	1 or less	m	III	1	N	N	N	N	N	N	N	T	N	Y	N	N	N	70	42	5	60	42	13	Nr	Nr	Nr	Nr	9	17.2	16	0.25	0.6	24	-	Y	Y	N	Y	N	12	S	I	1
3	806905	11mo	0.910	1 or less	m	III	-	N	N	N	N	N	N	N	T	AM	N	N	N	N	88	58	7	69	46	14	Nr	Nr	Nr	Nr	10	11.8	17	0.24	0.5	18	-	Y	Y	N	Y	N	13	M	I	2
4	882759	18y	18.000	15 to 18	m	I	3	N	N	N	N	N	N	N	T	N	N	N	N	N	110	62	43	165	-	-	Nr	Nr	Nr	Nr	7	13.5	20	1.1	1.25	28	-	N	N	N	N	N	8	S	I	1
5	885745	8y	8.000	6.1 to 12.0	m	II	2	N	N	N	N	N	N	N	T	N	N	N	N	N	100	60	18	118	-	-	Nr	Nr	Nr	Nr	14	13.5	19	0.6	0.9	28	0	Y	-	-	Y	N	14	M	O	1
6	891440	4y	4.000	3.1 to 6.0	m	II	3	N	N	N	N	N	N	N	T	N	N	N	N	N	94	62	17	100	42	14	Nr	Nr	Nr	Nr	7	10.7	18	0.86	0.8	22	-	N	N	N	N	N	8	S	I	-
7	892901	I.5y	1.500	1.1 to 3.0	m	I	-	N	Y	N	N	N	N	HDN	T	N	N	Y	N	N	80	42	9.8	77	48	14	Nr	Nr	Nr	Nr	4	7.4	17	0.23	0.55	20	-	N	Y	Y	Y	N	4	S	I	1
8	882431	1y	1.000	1 or less	m	II	1	N	N	N	N	N	N	N	T	N	Y	Y	N	N	90	50	7.4	57	43	13	Nr	Nr	Nr	Nr	5	10.8	14	0.53	0.88	18	-	Y	N	Y	N	N	6	S	I	1
9	891141	6mo	0.500	1 or less	f	III	-	N	N	N	N	N	N	N	T	AM	Y	Y	N	N	86	64	5.6	60	42	16	Nr	Nr	Nr	Nr	12	12	18	0.5	0.66	10	-	Y	Y	N	Y	N	1	M	I	-
10	882275	5mo	0.410	1 or less	m	I	1	N	N	N	N	N	N	N	T	N	N	N	N	N	90	60	6.7	54	45	10	Nr	Nr	Nr	Nr	8	13.2	19	0.8	0.99	30	-	Y	N	N	N	N	9	S	I	-
11	914314	2mo	0.160	1 or less	m	I	3	N	N	N	N	N	N	N	T	N	Y	N	N	N	65	50	8	60	37	-	Nr	Nr	Nr	Nr	7	9.7	19	0.24	0.55	15	-	N	N	N	N	N	8	S	I	1
12	880421	7y	7.000	6.1 to 12.0	m	III	3	N	N	N	N	N	N	N	T	N	Y	N	N	N	102	62	16	112	-	-	Nr	Nr	Nr	Nr	7	13.2	19	0.9	0.9	18	-	N	N	N	N	N	8	S	I	-
13	882086	3y	3.000	1.1 to 3.0	m	III	1	N	N	N	N	N	N	N	T	N	N	N	N	N	92	60	13	88	49	14	Nr	Nr	Nr	Nr	8	10.4	20	0.8	0.78	16	-	N	N	N	N	N	9	S	I	-
14	878731	3d	0.008	1 or less	m	III	-	N	N	N	N	N	N	HDN	T	MT,MPE	N	N	N	N	60	28	2.7	47	-	-	Abn	Nr	Nr	Nr	4	21.4	17	2.57	1.68	82	-	Y	Y	N	N	N	4	S	I	-
15	882304	8mo	0.660	1 or less	m	I	1	N	N	N	N	N	N	N	T	N	N	N	N	N	98	42	8	79	-	-	Nr	Nr	Nr	Nr	8	14	19	0.5	0.8	20	-	N	N	N	N	N	9	S	I	1
16	884054	5d	0.013	1 or less	m	III	1	N	N	N	N	N	N	PLS	T	N	N	N	N	Y	68	46	3	49	-	-	Nr	Nr	Nr	Nr	15	11.7	11	0.5	0.5	29	-	N	Y	N	N	N	15	B&U	O	-
17	882610	12y	12.000	6.1 to 12.0	m	I	1,2	N	N	N	N	N	N	N	T	N	N	N	N	N	110	70	33	144			Nr	Nr	Nr	Nr	5	13.1	30	0.8	1.5	27	-	Y	N	N	Y	N	4	B&U	O	2
18	882126	13y	13.000	12.1 to 15	m	I	1	N	N	N	N	N	N	N	T	N	N	N	N	N	100	60	49	150	-	-	Nr	Nr	Nr	Nr	8	12.1	19	0.7	1.4	16	-	N	N	N	N	N	9	S	I	2
19	890497	11mo	0.910	1 or less	m	II	2	N	N	N	N	N	N	AgK	Olig,AgK	T	AM	N	N	N	N	85	36	7.9	72	-	-	Nr	Nr	Abn	Nr	14	7.9	12	0.23	0.25	20	-	Y	Y	N	Y	N	1	B&U	O	-
20	881197	7y	7.000	6.1 to 12.0	m	II	1,2	N	N	N	N	N	N	N	T	N	N	N	Y	N	100	56	15	110	-	-	Nr	Nr	Nr	Nr	14	12.1	21	0.38	0.3	15	-	Y	Y	Y	Y	N	16	S	I	-
21	877479	3mo	0.008	1 or less	m	III	-	N	N	N	N	N	N	HDN	HDN	T	N	N	N	N	61	26	2.6	48	-	-	Nr	Nr	Nr	Nr	4	11.1	16	0.52	0.48	18	-	Y	N	N	N	N	4	M	O	-
22	5158201	3mo	0.008	1 or less	m	I	-	N	N	N	N	N	N	PLS	T	N	N	N	Y	N	64	28	2.8	50	-	-	Nr	Nr	Nr	Nr	15	-	14	0.5	0.5	40	-	Y	N	N	N	N	17	M	O	-
23	932687	2y	2.000	1.1 to 3.0	m	II	1,2	N	N	N	N	N	N	N	T	N	Y	N	N	N	80	60	9.2	79	47	14	Nr	Nr	Nr	Nr	5	11	16	0.42	0.44	-	-	Y	Y	N	Y	N	10	S	I	-
24	930560	11y	11.000	6.1 to 12.0	m	II	2	N	N	N	N	N	N	N	T	N	N	N	N	N	90	60	19	126	-	-	Nr	Nr	Nr	Nr	14	8	16	4.5	4.2	101	-	Y	N	N	Y	N	4	S	I	-
25	935443	3mo	0.008	1 or less	m	II	-	N	N	N	N	N	N	Olig	T	N	N	N	N	N	60	34	3.5	50	-	-	Nr	Nr	Nr	Nr	1	20.2	10	0.4	0.4	18	-	Y	N	N	N	N	3	B&U	O	-
26	932276	2.6y	2.600	1.1 to 3.0	m	II	3	N	N	N	N	N	N	N	T	N	N	N	N	N	74	50	12	102	48	14	Nr	Nr	Nr	Nr	7	9.7	18	0.4	0.35	26	-	N	N	N	N	N	8	S	I	-
27	933483	3y	3.000	1.1 to 3.0	m	II	3	N	N	N	N	N	N	N	T	N	N	N	N	N	76	52	14	99	50	16	Nr	Nr	Nr	Nr	7	11.2	19	0.6	0.4	24	-	N	N	N	N	N	8	S	I	-
28	923036	3.6mo	0.300	1 or less	m	II	1,4	N	N	N	N	N	N	DK,Olig	DK,Olig	T	N	Y	N	N	80	58	5.8	58	-	-	Nr	Nr	Nr	Nr	9	14.4	19	0.5	1	17	-	N	Y	N	N	N	12	S	O	2
29	914543	2y	2.000	1.1 to 3.0	m	III	1	N	N	N	N	N	N	N	T	AM	Y	Y	N	Y	94	68	8	81	46	14	Nr	Nr	Nr	Nr	5	7.7	14	0.3	0.4	15	-	N	Y	N	Y	N	18	S	I	-
30	913139	2mo	0.160	1 or less	m	II	1	N	N	N	N	N	N	N	HDN,Olig	T	N	Y	N	N	84	58	4.9	58	-	-	Nr	Nr	Nr	Nr	4	10.7	19	0.18	0.2	10	-	Y	Y	N	N	N	4	S	I	-
31	912143	2.6y	2.600	1.1 to 3.0	f	II	2	N	N	N	N	N	N	N	T	N	Y	N	N	N	98	62	15	90	42	14	Nr	Nr	Nr	Nr	14	11.3	16	0.21	0.2	18	-	Y	N	N	Y	Y	4	S	I	-
32	882762	4y	4.000	3.1 to 6.0	m	I	1	N	N	N	N	N	N	N	T	N	N	Y	N	Y	100	60	17	96	49	14	Nr	Nr	Nr	Nr	5	11.4	19	0.8	0.75	28	-	Y	Y	N	Y	N	10	S	I	-
33	885582	6y	6.000	3.1 to 6.0	m	II	3	N	N	N	N	N	N	N	T	N	N	Y	N	N	90	70	19	113	-	-	Nr	Nr	Nr	Nr	7	12.3	15	0.33	0.35	25	-	N	N	N	N	N	8	S	I	-
34	922801	1.6y	1.600	1.1 to 3.0	m	III	2	N	N	N	N	N	N	N	T	N	N	N	N	N	96	56	7.8	67	44	14	Nr	Nr	Nr	Nr	14	6.9	11	2.96	2.92	139	-	Y	Y	N	N	N	4	S	I	-
35	913523	20d	0.055	1 or less	m	III	1,2	N	N	N	N	N	N	N	T	N	N	N	N	N	60	36	3.2	50	-	-	Nr	Nr	Abn	Nr	4	14.2	14	2.37	2.52	92	-	Y	Y	N	Y	N	11	S	I	2
36	921499	1y	1.000	1 or less	m	III	1,3	N	N	N	N	N	N	N	N	P	N	N	N	N	90	56	8.5	80	45	14	Nr	Nr	Nr	Nr	7	12	19	0.2	0.25	29	-	N	N	N	N	N	8	S	I	-
37	923299	1y	1.000	1 or less	m	III	3	N	N	N	N	N	N	N	T	N	N	N	N	N	88	56	9.2	75	44	15	Nr	Nr	Nr	Nr	7	11.4	16	0.23	0.44	14	-	N	N	N	N	N	8	S	I	-
38	912510	1.2y	1.200	1.1 to 3.0	m	III	3	N	N	N	N	N	N	N	T	N	N	N	N	N	88	58	9.8	84	45	14	Nr	Nr	Nr	Nr	7	9.8	19	0.6	0.6	57	-	N	N	N	N	N	8	S	I	-
39	894995	3.8y	3.800	3.1 to 6.0	m	III	3	N	N	N	N	N	N	N	T	N	N	N	N	N	90	70	12	99	51	15	Nr	Nr	Nr	Nr	7	10.9	19	0.7	0.65	28	-	N	N	N	N	N	8	S	I	-
40	895096	7y	7.000	6.1 to 12.0	m	III	1	N	N	N	N	N	N	N	T	N	N	N	N	N	98	58	20	122	-	-	Nr	Nr	Nr	Nr	5	13.2	25	0.42	0.89	19	-	N	Y	N	Y	N	11	S	I	2
41	873709	6y	6.000	3.1 to 6.0	m	III	-	N	N	N	N	N	N	HDN	P	N	Y	N	N	N	96	62	14	110	-	-	Nr	Nr	Nr	Nr	4	9.9	20	1.21	1.56	33	-	Y	N	N	Y	N	11	M	I	1
42	885920	4mo	0.011	1 or less	f	III	-	N	N	N	N	N	N	N	T	N	N	N	N	N	60	36	2.2	48	-	-	Nr	Nr	Nr	Nr	13	17.2	14	0.8	0.78	37	-	Y	N	N	N	N	19	B&U	O	-
43	914442	13y	13.000	12.1 to 15	m	III	1	N	N	N	N	N	N	N	T	N	N	N	N	N	120	84	30	146	-	-	Nr	Nr	Nr	Nr	5	11.8	17	0.63	0.6	22	-	Y	N	N	Y	N	4	S	I	-
44	915132	1y	1.000	1 or less	m	III	-	N	N	N	N	N	N	N	T	N	N	N	N	N	86	56	9	74	44	14	Nr	Nr	Nr	Nr	4	9.9	16	1	0.98	24	-	Y	N	N	N	N	4	S	I	-
45	915194	5mo	0.410	1 or less	m	III	3	N	N	N	N	N	N	N	T	N	Y	N	Y	N	80	50	6	62	-	-	Nr	Nr	Nr	Nr	7	12	17	0.5	0.6	16	-	Y	Y	N	Y	N	20	S	I	-

46	915175	3mo	0.008	1 or less	m	I	-	N	N	N	N	N	N	AgK	T	N	N	N	N	Y	66	54	2.6	50	-	-	Nr	Nr	Nr	Nr	15	11.1	16	0.4	0.4	20	-	Y	N	N	Y	N	1	B&U	O	-
47	934902	5mo	0.014	1 or less	m	III	-	N	N	N	N	N	N	N	T	N	N	N	N	N	64	52	2.8	50	-	-	Nr	Nr	Nr	Nr	4	15.2	17	0.32	0.4	18	-	Y	N	N	N	N	4	M	I	-
48	927644	1.6mo	0.130	1 or less	m	III	2	N	N	N	N	N	N	N	T	CHD,VSD	N	N	N	N	70	52	3.5	52	-	-	Nr	Nr	Nr	Nr	15	13	10	3.35	3.2	123	-	Y	Y	N	Y	N	10	M	O	-
49	892911	5y	5.000	3.1 to 6.0	m	I	2	N	N	N	N	N	N	N	T	N	N	N	N	N	92	56	17	117	-	-	Nr	Nr	Nr	Nr	15	11	14	0.36	0.35	13	-	Y	Y	Y	Y	N	10	S	I	-
50	894994	6mo	0.500	1 or less	m	IV	1	N	N	N	N	N	N	N	T	N	N	N	N	N	72	54	5.6	57	41	12	Nr	Nr	Abn	Nr	5	8.6	21	1	0.98	31	-	Y	Y	N	N	N	21	S	I	-
51	913139	2mo	0.160	1 or less	m	III	1,2	N	N	N	N	N	N	HDN,Olig	T	N	N	N	N	N	74	54	4.8	58	-	-	Nr	Nr	Nr	Nr	15	12.3	14	2.51	2.2	73	-	Y	Y	N	N	N	11	S	I	-
52	920871	3mo	0.008	1 or less	m	III	-	N	N	N	N	N	N	DK	T	N	N	N	N	N	60	36	3.2	52	-	-	Nr	Nr	Nr	Nr	6	20.8	7	1.28	1.2	99	-	Y	N	N	N	N	7	B&U	O	-
53	933784	4mo	0.011	1 or less	m	III	-	N	N	N	N	N	N	HDN	P	N	N	Y	Y	Y	38	22	1.1	40	-	-	Nr	Nr	Nr	Nr	4	20.3	16	0.66	0.58	17	-	Y	N	N	N	N	4	M	O	-
54	894555	1.5mo	0.125	1 or less	m	I	1	N	N	N	N	N	N	N	T	N	N	N	N	N	88	60	7	74	44	11	Nr	Nr	Nr	Nr	7	11	12	0.6	0.55	20	-	N	N	N	N	N	8	S	I	-
55	4588270	11mo	0.910	1 or less	m	III	1	N	N	N	N	N	N	N	T	N	Y	N	N	N	90	58	8.6	70		-	Nr	Nr	Nr	Nr	5				0.42		-	Y	Y	N	Y	N	22	S	I	-
56	892434	4.8mo	0.330	1 or less	m	II	2	N	N	N	N	N	N	N	T	N	Y	N	N	N	96	54	16	102	48	15	Nr	Nr	Nr	Nr	14	11.6	22	0.42	0.4	23	-	Y	Y	N	Y	N	11	S	I	-
57	923869	9mo	0.750	1 or less	f	I	-	N	N	N	N	N	DK	DK	T	N	N	Y	Y	N	80	58	6.5	64	-	-	Nr	Nr	Nr	Nr	6	11.4	15	0.21	0.2	23	-	Y	Y	N	Y	N	7	B&U	O	-
58	916656	11mo	0.910	1 or less	m	II	2,4	N	N	N	N	N	HDN	HDN	T	N	N	N	N	N	90	58	7.8	68	-	-	Nr	Nr	Nr	Nr	14	10.2	17	0.19	0.2	-	-	Y	N	N	N	N	4	S	I	-
59	93328	3mo	0.008	1 or less	f	III	-	N	N	N	N	N	N	Olig	T	N	N	N	N	N	60	32	3	50	-	-	Nr	Nr	Nr	Nr	1	-	10	0.75	0.72	39	-	Y	N	N	N	N	1	B&U	O	-
60	963714	20d	0.055	1 or less	m	III	1,4	N	N	N	N	N	N	Olig	T	N	N	N	N	N	80	50	2.7	50	-	-	Nr	Nr	Nr	Nr	4	-	19	0.6	1.2	56	-	Y	Y	N	Y	N	11	S	I	2
61	924532	4mo	0.011	1 or less	f	III	-	N	N	N	N	N	N	DK,Olig	T	N	N	N	Y	N	60	38	2.5	50	-	-	Nr	Nr	Nr	Nr	3	19	18	0.49	0.5	30	-	Y	N	N	N	N	3	B&U	O	-
62	875396	13y	13.000	12.1 to 15	m	III	-	N	N	N	N	N	N	N	T	N	N	N	N	N	108	74	32	149	-	-	Nr	Nr	Nr	Nr	1	11.9	19	0.94	0.9	22	-	Y	N	N	N	N	1	B&U	O	-
63	892595	1y	1.000	1 or less	m	III	2	N	N	N	N	N	N	N	T	N	N	N	N	N	85	52	8	80	46	13	Nr	Nr	Nr	Nr	14	7.2	14	0.18	0.25	21	-	Y	N	Y	Y	N	4	S	I	-
64	873176	11y	11.000	6.1 to 12.0	m	III	1	N	N	N	N	N	N	N	T	N	N	N	N	N	92	60	31	145	-	-	Nr	Nr	Nr	Nr	7	11.6	18	0.9	0.8	27	-	Y	N	N	N	N	1	B&U	O	-
65	874736	8d	0.022	1 or less	m	III	-	N	N	N	N	N	N	Olig	T	N	N	N	N	N	54	28	3.4	47	-	-	Nr	Nr	Nr	Nr	4	17.6	15	1.14	1.09	52	-	Y	N	N	N	N	4		O	-
66	885535	1mo	0.080	1 or less	m	III	1,4	N	N	N	N	N	N	DK	T	N	N	N	N	N	60	38	4	50	-	-	Nr	Nr	Nr	Nr	5	16.5	15	1.01	0.9	23	-	Y	Y	N	N	N	23	S	I	-
67	889503	2y	2.000	1.1 to 3.0	m	III	1	N	N	N	N	N	N	N	T	N	Y	N	N	N	80	56	9.5	80	46	14	Nr	Nr	Nr	Nr	7	13.5	15	0.8	0.77	22	-	Y	Y	N	N	N	24	S	I	-
68	923007	9y	9.000	6.1 to 12.0	m	III	2	N	N	N	N	N	N	N	T	N	N	N	Y	N	106	60	18	119	-	-	Nr	Nr	Nr	Nr	14	11.9	19	0.45	0.5	28	-	Y	N	N	Y	N	4	S	I	-
69	893798	1.6y	1.600	1.1 to 3.0	m	III	3	N	N	N	N	N	N	N	T	N	N	N	N	N	84	54	8.5	76	-	-	Nr	Nr	Nr	Nr	7	11.7	19	0.8	0.78	15	-	N	N	N	N	N	8	S	I	-
70	923250	4y	4.000	3.1 to 6.0	m	IV	3	N	N	N	N	N	N	N	T	N	N	N	N	N	90	50	15	101	48	14	Nr	Nr	Nr	Nr	7	12	18	0.6	0.55	18	-	N	N	N	N	N	8	S	I	-
71	932695	1.2y	1.200	1.1 to 3.0	m	III	3	N	N	N	N	N	N	N	T	N	N	N	N	N	70	38	9.5	78	47	15	Nr	Nr	Nr	Nr	7	13.1	18	0.5	0.44	20	-	N	N	N	N	N	8	S	I	-
72	932626	11y	11.000	6.1 to 12.0	m	III	2	N	N	N	N	N	N	N	T	N	N	N	N	N	102	58	32	135	-	-	Nr	Nr	Nr	Nr	14	12.8	18	0.6	0.45	22	-	N	N	Y	Y	N	4	B&U	O	-
73	965185	4mo	0.011	1 or less	m	III	-	N	N	N	N	N	N	Olig	T	N	N	N	N	N	62	36	9	2.8	50	-	Nr	Nr	Nr	Nr	4	16	10	0.61	0.6	19	-	Y	Y	N	N	N	4	B&U	O	-
74	965162	4mo	0.011	1 or less	f	III	-	N	N	N	N	N	N	Olig	T	N	N	N	Y	N	60	38	3.5	50	-	-	Nr	Nr	Nr	Nr	11	16	17	0.4	0.32	22	-	Y	N	N	N	N	15	B&U	O	-
75	965186	4mo	0.011	1 or less	m	III	-	N	N	N	N	N	N	Olig	T	N	N	N	N	N	64	36	3.3	49	-	-	Nr	Nr	Nr	Nr	2	15.5	14	0.45	0.52	20	-	Y	N	N	N	N	2	B&U	O	-